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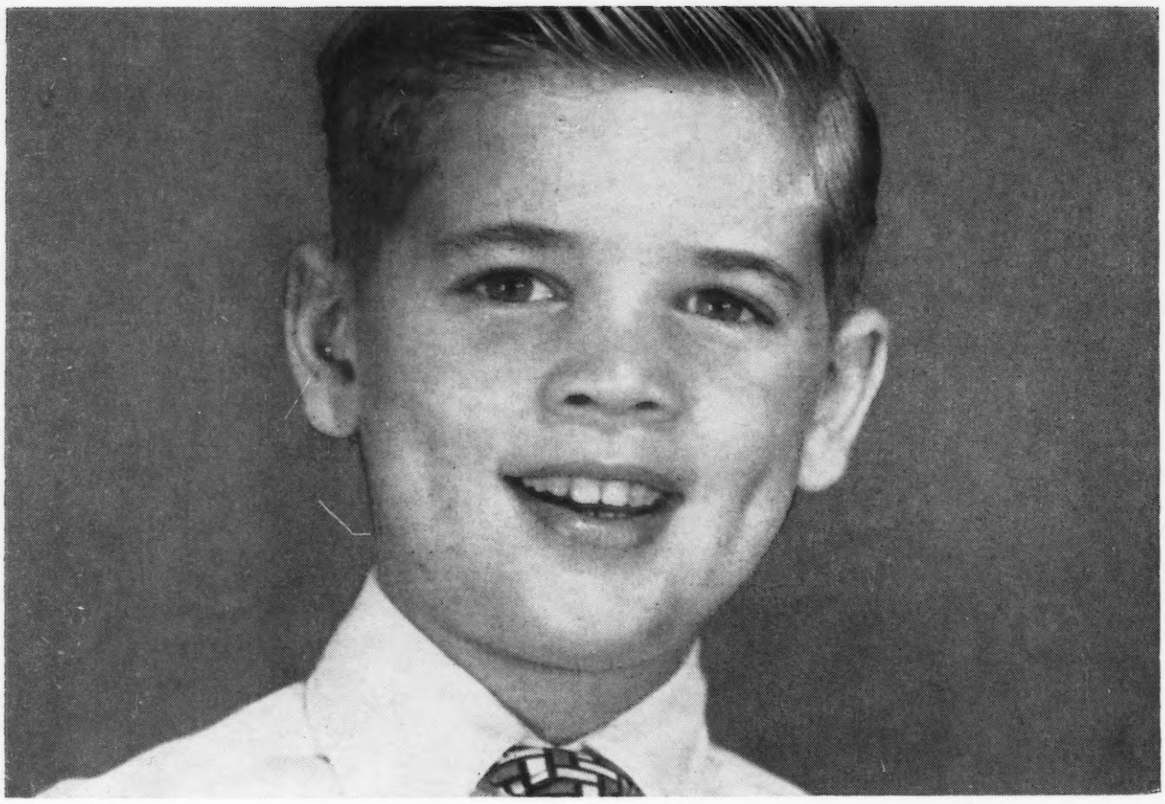

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1. Means, J.H.: The Thyroid and Its Diseases, ed. 2. Philadelphia, J.B. Lippincott Co., 1948.

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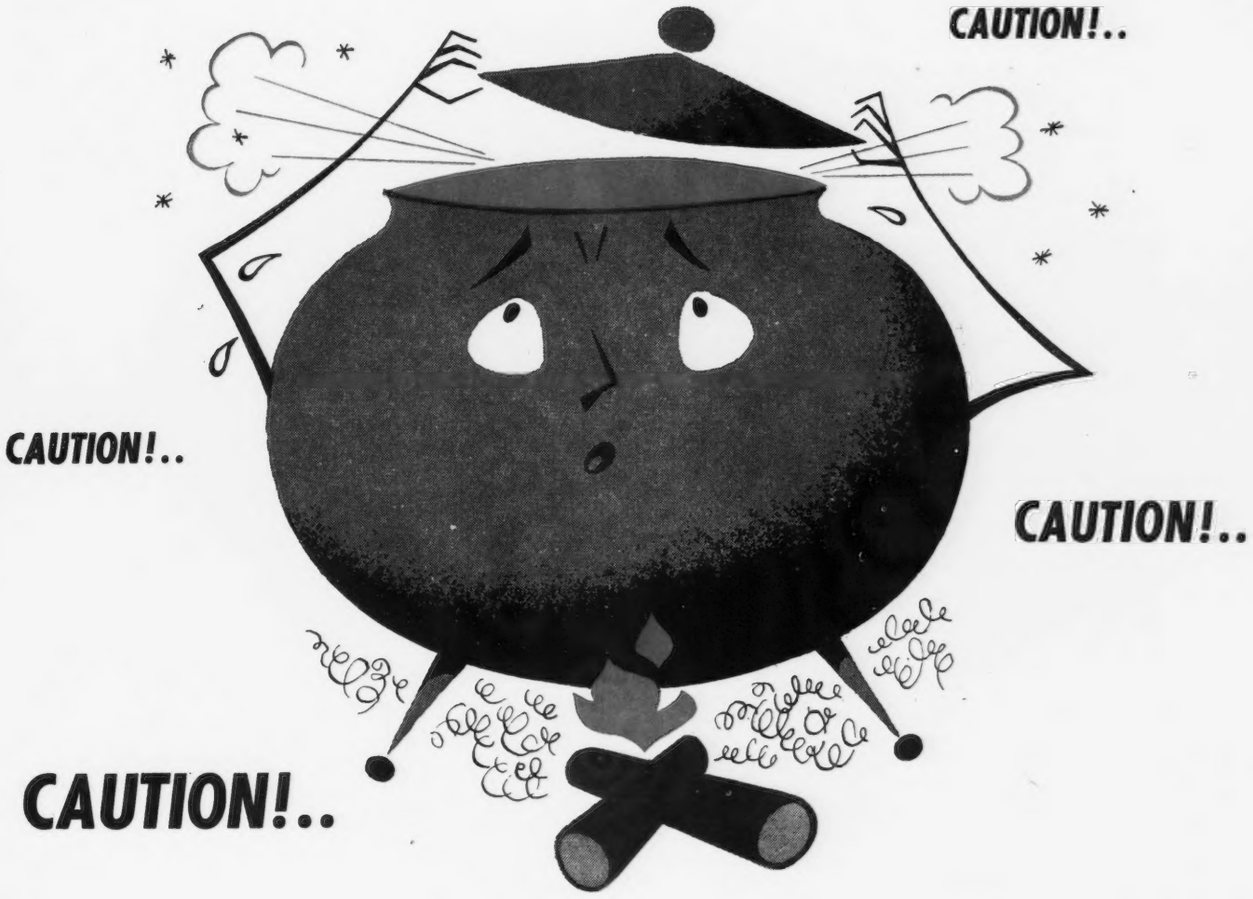

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REFERENCE: 1. Silcox, L. E., *A.M.A. Arch. Otolaryng.* **60**:431, Oct. 1954.

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Canad. M. A. J.
Mar. 1, 1956, vol. 74

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From The Journal of March, 1926

(FROM AN ARTICLE ON RABIES BY R. H. MALONE, M.D.)

Rabies was probably introduced into Canada in 1810, when there was a severe outbreak in Ohio. In 1820, the Duke of Richmond, then Governor-General of Canada, died of hydrophobia from the bite of a captive fox. In 1904, a little dog trotted over the suspension bridge at Queenston from New York State, bit a number of dogs, cattle and sheep, and trotted back again. Since then rabies has remained in Canada. It has spread from Ontario to Manitoba, Saskatchewan and Alberta. Cases have been reported in British Columbia too. Quebec seems to have remained free until this last outbreak.

QUEBEC NEWS

Among the portraits of the makers of McGill University's history that grace the walls of the Assembly Hall in the Medical Building, has recently been placed the portrait of Dr. Francis J. Shepherd, for many years Dean of the Medical Faculty. This painting is the work of Miss Gertrude Desclayes, and is a gift to the University from the many admirers of Dr. Shepherd, but chiefly from his former housemen and students. Dr. H. S. Birkett presented the painting to the University, saying that in doing so his students were paying respect to a beloved teacher whose widespread reputation in anatomy, surgery and dermatology had done much to make the name of McGill famous in many parts of the world.

[Canad. M. A. J.
March 1, 1956, Vol. 74

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Each tablet contains:

Sulfamethazine..... 167 mg.
Sulfadiazine..... 167 mg.
Sulfamerazine..... 167 mg.
} 7½ gr.

Crystalline potassium penicillin-G...
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Each 5 cc. teaspoonful contains:

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Sulfamerazine..... 167 mg.
} 7½ gr.

Benzathine penicillin-G.....
100,000 I.U. — "TRULFACILLIN" 7½-100
150,000 I.U. — "TRULFACILLIN" 7½-150
300,000 I.U. — "TRULFACILLIN" 7½-300

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SUSPENSIONS

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Sulfadiazine..... 65 mg.
Sulfamerazine..... 65 mg.
} 3 gr.

Benzathine penicillin-G.....
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While untoward effects associated with sulfonamide therapy are greatly reduced by the use of Trulfacillin preparations, vigilance should not be relaxed in the search for and recognition of agranulocytosis, fever, joint pains, skin reactions, etc. In rare instances the injection of penicillin, and more rarely still its oral administration, may cause acute anaphylaxis. The reaction appears to occur more frequently in patients with bronchial asthma and other allergies, or in those who have previously demonstrated sensitivity to penicillin.

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[1]Lehr, David, "Present Status of Sulfonamide Therapy", Scientific Exhibit, Annual Convention A.M.A., San Francisco, 1954.

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Sulfamerazine.....	167 mg.	

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Each scored tablet contains:

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Sulfamerazine.....	167 mg.	

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SUSPENSION

Similar in formula to Trulfa-Zine Suspension, except that sulfathiazole is used instead of sulfamethazine.

TABLET

Similar in formula to Trulfa-Zine Tablet, except that sulfathiazole is used instead of sulfamethazine.

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SUSPENSIONS

Infants and Children: ½ teaspoonful (2.5 cc.) for each 4 pounds of body weight per day in divided doses (approximately 1 grain per pound of body weight) e.g. Child's weight—24 pounds: 1 teaspoonful 3 times daily.
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CAUTION

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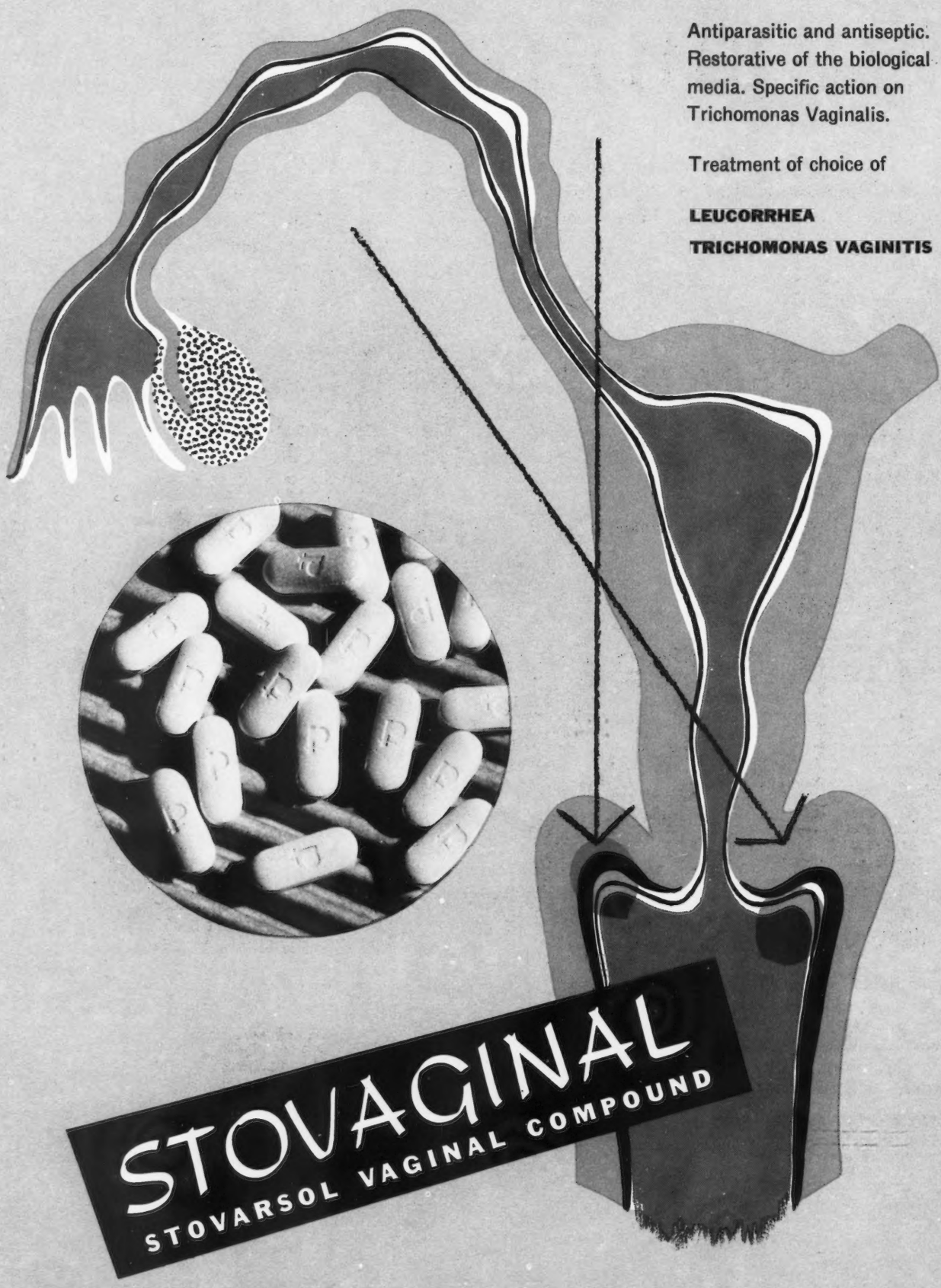
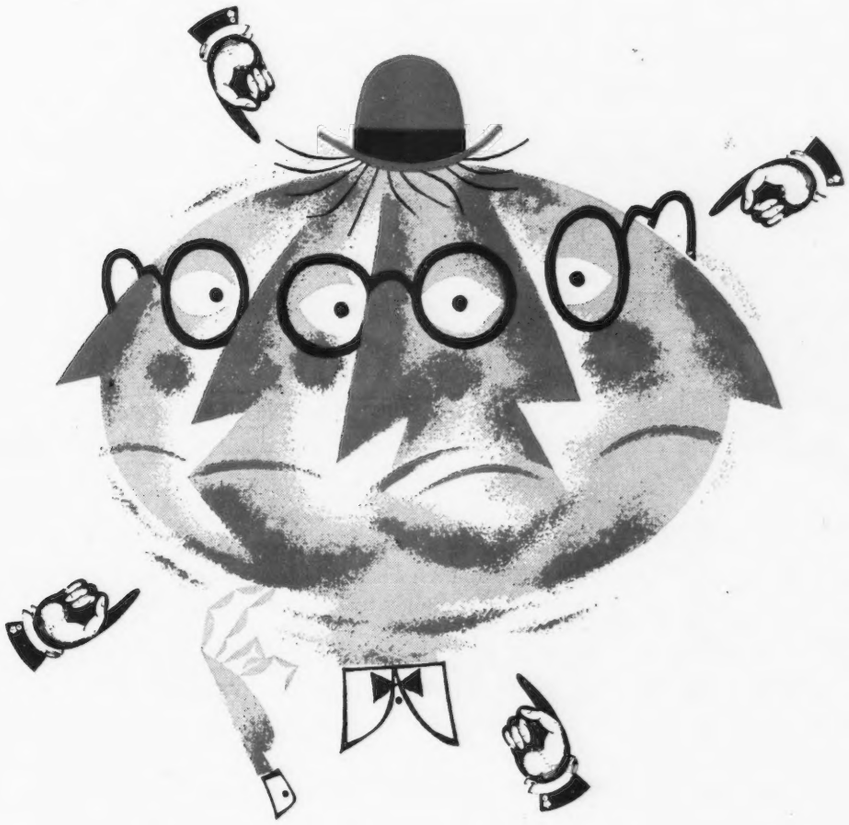


STOVAGINAL

STOVARSOL VAGINAL COMPOUND



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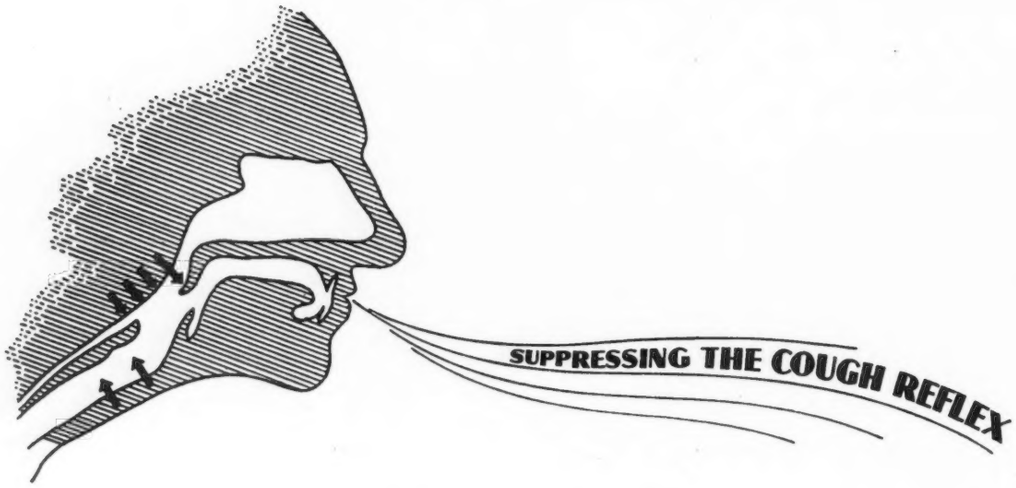

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Erythromycin in treatment of abscess

6/21/55

DISCHARGE SUMMARY

On 5/23/55 this patient (colored female, age 24) underwent an excisional biopsy of a breast tumor. On 5/24 tumor was removed and patient discharged from hospital on following day.

On 6/3/55 patient was readmitted because of purulent discharge from wound. On 6/3 a hemolytic Staph. aureus (coag. +) was isolated from abscess with the following disk sensitivities: penicillin, 1.5 units; erythromycin, 10 mcg; tetracycline, 10 mcg. Patient was placed on penicillin, 600,000 units b.i.d. for 10 days. On this schedule patient improved but progress was unsatisfactory and wound continued to discharge small amount of purulent material.

On 6/13 penicillin was discontinued and erythromycin* started in dosage of 200 mgm. q.i.d. By 6/17 the discharge had stopped and wound was completely healed by 6/19. Erythromycin was continued until the patient was discharged from hospital on 6/21. Temp. was normal throughout hospital stay.

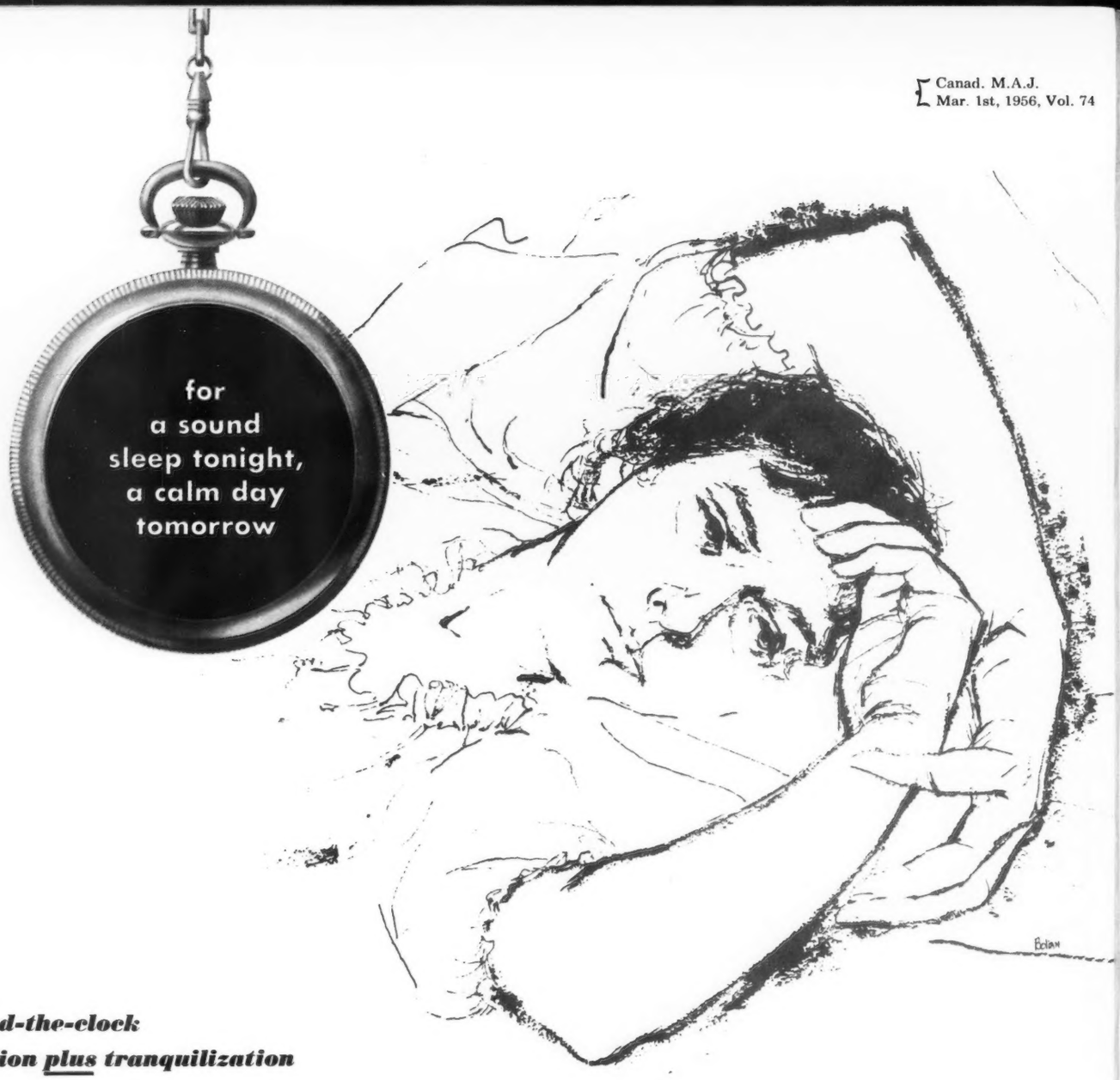
Final diagnosis: breast abscess due to Staph. aureus.

Result: rapid and complete recovery on erythromycin following failure of penicillin.

Communication to Abbott Laboratories

*ERYTHROCIN (Abbott)

Canad. M.A.J.
Mar. 1st, 1956, Vol. 74



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MARCH 1, 1956 • VOL. 74, NO. 5

VALUE OF NEWER TISSUE CULTURE METHODS IN EPIDEMIOLOGICAL INQUIRIES: AN ILLUSTRATIVE OUTBREAK OF POLIOMYELITIS*

A. J. BEALE, M.D., Dip. Bact.(Lond.),
M. W. FUJIWARA, B.A., M.D.,
W. STACKIW, B.Sc.,
NORMA DAVIS, B.A. and
A. J. RHODES, M.D., F.R.C.P.(Edin.),
Toronto

THE STUDY OF POLIOMYELITIS has been enormously facilitated by the discovery of Enders, Weller and Robbins[9] that the virus will grow in tissue cultures. This technical advance has been widely used for virus isolation and diagnosis, serological investigation, and vaccine production.[8, 19, 22] As yet, however, relatively few outbreaks have been investigated epidemiologically by means of these techniques.[2-4, 11, 12, 23]

Prior to the introduction of tissue cultures, the only method of isolating poliomyelitis virus was the time-consuming and expensive one of inoculating monkeys. Consequently, investigations into the manner of spread of infection depended largely on the validity of the clinical diagnosis of poliomyelitis. The clinical diagnosis of the ambulant and non-paralytic form is, however, notoriously inaccurate. Undoubtedly, in the past, Coxsackie and other infections have been erroneously ascribed to poliomyelitis.[5, 18] In earlier studies, investigators have rarely had the opportunity to assign the poliomyelitis strains isolated to one of the three standard types. This was a serious drawback, and in a study reported by our group some years ago, a child (R.A.) was incriminated as the probable introducer of infection into a small Ontario country town, on the basis of epidemiological evidence supported by virus isolations.[21] Typing of the strains then isolated by the new methods has shown that R.A. was, in fact, infected with Type 2 virus, whereas the other victims were all infected with Type 1. Clearly, R.A. was wrongly blamed and the real source of the outbreak was not traced.

Tissue cultures, especially trypsinized monkey-kidney cultures, afford the means of overcoming most of the difficulties encountered in earlier studies. Culture tubes can be prepared readily and in quantity, and are very sensitive to poliomyelitis, as well as Coxsackie B and "orphan" viruses. Since, in Canada, Type 1 strains cause about 90% of clinical poliomyelitis, finer serological methods of characterizing the antigenic structure of particular epidemic strains would be of great value in tracing the spread of infection.[7] Such methods are not yet available.

The present paper describes a poliomyelitis outbreak which occurred in April 1955 in Toronto. The new tissue-culture methods were used as an adjunct to the conventional methods of epidemiological inquiry. The outbreak is of interest because it appeared to be initiated by an infected resident recently returned from Mexico. Examples of such introduction of infection have more commonly been described in remote communities.[15, 16] It is also of interest that although the strain was highly communicable and invasive, the outbreak terminated within the space of only a few transfers.

METHODS

Trypsinized monkey-kidney monolayer cultures were prepared by Melnick's modification[13] of the methods introduced by Dulbecco and Vogt[6] and Youngner.[24] Epithelial sheets were grown in Synthetic Medium No. 199 containing 1-2% horse serum;[14] at the time of infection, the nutrient was changed to Medium 199 (without serum). Penicillin (500 units/ml.) and streptomycin (250 micrograms/ml.) were added to all media.

ISOLATION OF VIRUS

A suspension of fæces (10-20%) was made in Hanks' balanced salt solution by shaking with glass beads. The

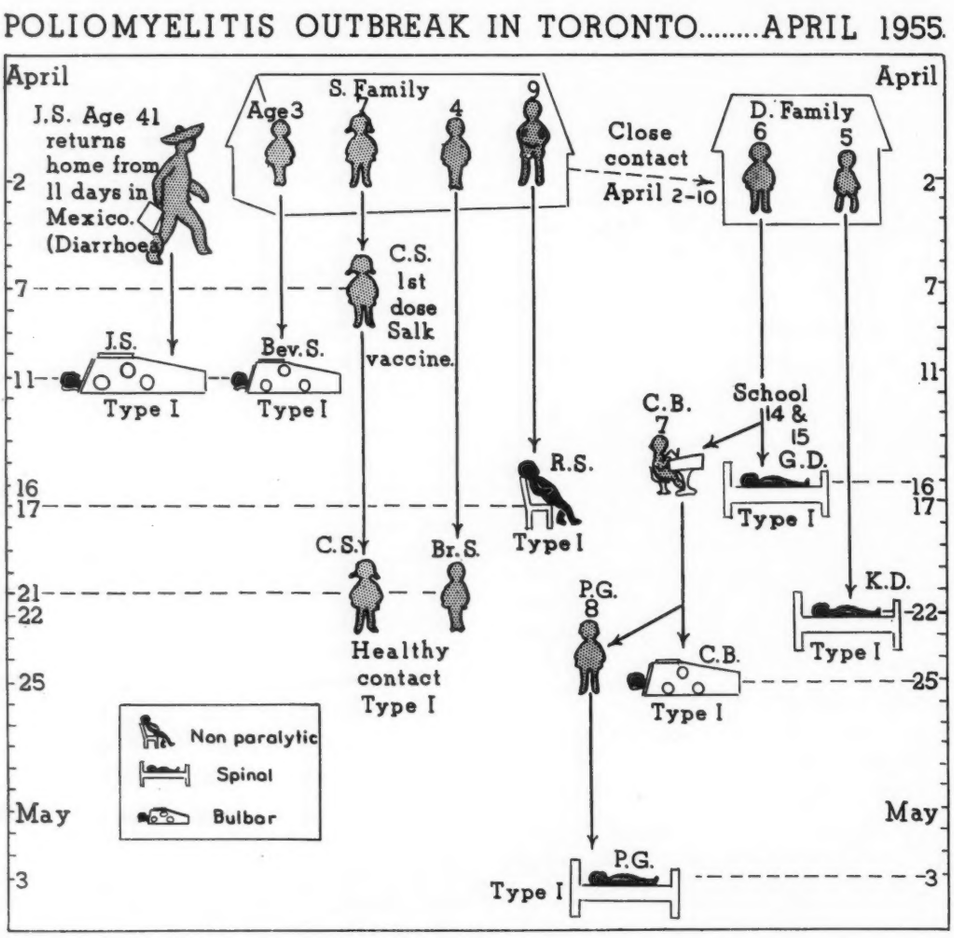
*From the Department of Pædiatrics, University of Toronto, and the Research Institute, the Hospital for Sick Children, Toronto, Ontario.
Assisted with funds allocated by the Province of Ontario under the National Health Grants Programme of the Department of National Health and Welfare, Ottawa.

suspension was centrifuged in a Spinco ultracentrifuge at 15,000 r.p.m. for 30 minutes; the supernatant fluid, after the addition of penicillin (500 units/ml.) and streptomycin (250 micrograms/ml.), was inoculated into well-grown monkey-kidney monolayer tissue cultures. Rectal swabs were placed in 1 ml. of Hanks' balanced salt solution containing antibiotics, and these suspensions were used to inoculate tissue cultures.

Cultures were examined daily under the low power of the microscope for cytopathogenic changes. Cultures in which the cells showed no such changes were kept for one week, and were then subcultured into another series. Cultures showing cytopathogenic changes were subjected to examination by "typing";[7] tissue-culture fluids suspected to contain virus were mixed with equal volumes of hyperimmune monkey antisera prepared for the three serological types of poliomyelitis virus. After the mixtures had stood for one hour at room temperature, groups of cultures were inoculated. It was often possible to read the preliminary results after 24-48 hours, but the cultures were left for one week before the final readings were made.

SEROLOGICAL TESTS

Neutralizing antibody titrations were performed by the pH colour test introduced by Salk *et al.*[20] Serial twofold dilutions of serum were made in 0.25 ml. volumes, and an equal volume of 1:100 suspension of trypsinized monkey-kidney cells was added. One hundred CPD_{50} of strains of each type of poliomyelitis virus in 0.25 ml. were then added, and the tubes were sealed with a layer of sterile mineral oil.[25] The results were read after 10 days, by observing pH changes and examining microscopically for the presence or absence of cells. Each test included, in addition to a serum control, a cell, virus, and gamma globulin titration, to check the reproducibility and sensitivity of the tests. Acute and convalescent phase sera from each patient were tested at the same time. The antibody titre was expressed as the initial dilution of serum in the last tube showing complete protection of the cells by the serum against the cytopathogenic effect of the virus.



DESCRIPTION OF THE OUTBREAK

The sequence of events is illustrated in the figure, and the clinical features are outlined in Table I. J.S., aged 41, returned from a trip to Mexico on April 2, 1955. He was feeling unwell and had mild diarrhœa, but went to work on April 4. He fell ill again on April 11 with fever, drowsiness, headache, and vomiting. Difficulty in swallowing developed the next day, and on April 13 he was admitted to hospital. Later, he developed weakness of his shoulders. His youngest daughter Bev.S., aged 3 years, also fell ill

on April 11 with similar symptoms and was admitted to hospital on April 13. She later developed a lower motor neurone facial palsy.

The next patient was R.S., aged 9, who developed non-paralytic poliomyelitis on April 17. The other children in the "S" family, C.S. aged 7, and Br.S. aged 4, remained healthy. Rectal swabs were taken on April 21. C.S. had received her first dose of poliomyelitis vaccine ("Salk type") on April 7.

TABLE I.

POLIOMYELITIS OUTBREAK IN TORONTO, 1955: CLINICAL FEATURES

	Date of onset				
Initials	*Minor illness*	*Major illness*	*Quarantine imposed on family*	*C.S.F. findings*	*Type of disease*
J.S.	2/4/55	11/4/55	14/4	350 cells, mainly lymphocytes	Bulbospinal
Bev.S.	None	11/4		108 cells, all lymphocytes	Bulbar
R.S.	None	17/4		51 cells, mainly lymphocytes	Non-paralytic
G.D.	16/4	19/4	19/4	300 cells, mainly polymorphs	Spinal
K.D.	None	22/4		34 cells, 80% lymphocytes	Spinal
C.B.	None	25/4	28/4	90 cells, 80% lymphocytes	Bulbospinal
P.G.	26/4	2/5	3/5	300 cells, 90% lymphocytes	Spinal

The "D" family lived opposite, and their two children at home, G.D. (aged 6) and K.D. (aged 5), were constantly in the S. garden playing and having meals during the Easter holiday period (April 2 to 10). On April 14, G.D. returned to the private school where she was a day pupil. This school draws pupils from a wide area of Toronto. On April 16, she was feverish, but recovered in the next two days, though she did not return to school. On April 19 she was admitted to hospital with preparalytic poliomyelitis; there were 300 cells per c.mm. in the C.S.F. Later, left triceps weakness developed. On April 22, K.D. fell ill with a similar illness; there were 34 cells per c.mm. in the C.S.F. Weakness of the intercostals developed later.

C.B. (aged 7), a classmate of G.D., who had only been in contact with her on April 14 and 15, and lived some miles away, fell ill on April 25 with bulbar poliomyelitis. The last case in the outbreak was P.G., aged 8, who was a neighbour and friend of C.B., but did not attend the same school. She fell ill with mild paralytic poliomyelitis on May 3. This child had been given gamma globulin on April 30.

Routine quarantine measures were taken against the families of the cases of poliomyelitis as soon as the case was reported to the health authorities. The dates are shown in Table I. These measures consisted in "placarding" and confining all members of the family to the house for seven days from the time of their last contact with the clinical case. The development of poliomyelitis in C.B., who had been in contact with G.D. at school, raised a serious problem. The children in the class were excluded from school and many received gamma globulin on the recommendation of their pædiatricians. No more cases occurred in the school, and only one more clinical case was reported in the whole of Toronto during the ensuing month. This patient had no apparent connection with the outbreak now described and lived some miles away.

VIRUS STUDIES ON PATIENTS AND CONTACTS

Stools were obtained from all the seven patients, and all yielded Type 1 poliomyelitis virus, as shown in Table II. In addition, Type 1 poliomyelitis virus was isolated from the two healthy "S" children, Br.S. aged 4 and C.S. aged 7.

Table II also shows the serological results. It will be seen that four patients (J.S., R.S., C.B., and P.G.) had fourfold or greater rises in antibody to the type of virus isolated from their stools. Two cases (Bev.S. and G.D.) had equally high antibody levels to the infecting type of virus in acute and convalescent sera. One of these cases (C.B.) showed a rise in Type 2 antibodies, and another (P.G.) a rise to Type 3. Only one sample was taken from the remaining individuals and all showed a high antibody level to the infecting Type 1 strain. It is interesting that both adults had titres of over 1:16 against all 3 types of virus, whereas the children, although they had high titres to the causal Type 1 strain, had only low titres against Types 2 or 3.

TABLE II.

POLIOMYELITIS OUTBREAK IN TORONTO, 1955:
VIRUS ISOLATIONS AND POLIOMYELITIS NEUTRALIZING ANTIBODY LEVELS

Initials	*Age years*	*Type of disease*	*Serum collection (days after onset)*		*Neutralizing antibody titre to following** types of poliomyelitis virus*						*Poliomyelitis virus isolated from stool*
					1		*2*		*3*		
			*S1**	*S2**	*S1*	*S2*	*S1*	*S2*	*S1*	*S2*	
J.S.	41	Paralytic	4	23	64	1024	16	16	64	64	Type 1
Bev.S.	3	Paralytic	2	20	256	256	>2	>2	>2	>2	Type 1
R.S.	9	Non-paralytic	3	17	16	64	>4	>4	>4	>4	Type 1
G.D.	6	Paralytic	1	35	256	256	>4	>4	>4	>4	Type 1
K.D.	5	Paralytic	6	33	—	256	—	>2	—	>2	Type 1
C.B.	7	Paralytic	4	4 mos.	128	4096	>2	32	>2	>2	Type 1
P.G.	8	Paralytic	3	4 mos.	512	8192	8	8	>2	8	Type 1
C.S.	7	Healthy		19	—	256	—	4	—	>4	Type 1
Br.S.	4	Healthy		19	—	256	—	4	—	8	Type 1
Mrs.S.	—	Healthy		19	—	256	—	64	—	64	Not tested

*S1 = Acute and S2 = convalescent sample.
**Initial serum dilution.

DISCUSSION

The usefulness of tissue-culture methods in confirming the clinical diagnosis of poliomyelitis is once again demonstrated. The virus was readily isolated from the stools of all seven patients, in addition to two contacts. The first two cases were not at the outset typical clinically and, in view of the rarity of poliomyelitis in Toronto as early in the year as April, the laboratory confirmation of the diagnosis was valuable. In our experience, with trypsinized monkey-kidney cultures, virus can be isolated from nearly every case of clinically typical paralytic poliomyelitis. Similarly, other workers have found it possible to confirm the diagnosis in the laboratory in about 90% of paralytic cases.[8, 10]

Four of the six patients from whom two phase serum samples were obtained showed a four-fold or greater rise in antibody titre (Table II), which would be sufficient to establish the diagnosis even in the absence of virus isolation. Two patients had as high a titre in the acute as in the convalescent sample, so that the serological results alone did not establish a diagnosis of recent poliomyelitis infection. The occurrence of such antibody patterns illustrates that virus isolation is a more sensitive method of diagnosis, and is of course much faster.

The spread of infection in this outbreak could be postulated by observing clinical cases, and this was amply confirmed by the virus isolation tests. All the six children in the "S" and "D" families became infected with virus, and four suffered from clinical poliomyelitis, illustrating both the communicability and invasiveness of the Type 1 strain involved.

The origin of the outbreak is suggestive but not proven. In fact, since J.S. and Bev.S. became ill on the same day, it might be supposed that they both became infected at the same time, soon after J.S.'s return from Mexico. However, the absence of clinical poliomyelitis in Toronto, before or for a month after this outbreak, makes the alternative hypothesis that the virus was in fact imported from Mexico by J.S. very attractive. If this is correct, J.S. almost immediately on return from Mexico infected Bev.S., who developed symptoms after an incubation of not more than nine days.

The virus spread readily amongst the "S" and "D" children in their homes, yet by contrast appeared unable to spread amongst the school children of similar social background, and presumably therefore of similar susceptibility. In the "S" and "D" families, all the children were infected and 4 out of 6 had clinical manifestations. It is well known that there is a high infection rate amongst the family contacts of cases of poliomyelitis, and that such family contacts show a higher rate of clinically apparent infections than the general population.[3, 4, 12] For similar reasons, multiple clinical cases are common in nursery school outbreaks, whereas they

are rare in day schools of the type involved in this outbreak.[17]

It is often assumed that widespread subclinical infection none the less does take place in day schools, but there is very little direct evidence available. In a school outbreak in New York State recently reported by Winsser,[23] there were nine cases amongst 85 public school children all caused by Type 1 virus. Nine days after the last case, 300 stools or anal swabs were obtained from the school children and from home contacts. Only two poliomyelitis viruses (both Type 1) were isolated from these healthy contacts. The sampling may have been done too late to detect subclinical infection, or alternatively the contact at school may not have been close enough to spread the infection. This latter explanation seems more probable since it is well established that virus may be excreted for four or more weeks in the stool.

In view of the recent recommendation of stricter quarantine measures in the prevention of the spread of poliomyelitis,[1, 11,22] it is of interest that virus was recovered from Br.S. and C.S. just as the quarantine restrictions were lifted on the "S" family. As is customary in North America, the quarantine period was for seven days and applied only to the family associates, and not to close extra-familial contacts.

The dates on which quarantine was imposed on the families are shown in Table I. It is clear (Fig. 1) that the "D" family was already infected at the time the "S" family was quarantined on April 14. G.D. became ill two days later, and the family was put in quarantine on April 19. If both the "S" and "D" families had been placed in quarantine for three weeks on April 13, the day J.S. was admitted to hospital, as recommended by the World Health Organization Expert Committee on Poliomyelitis,[22] it seems probable that both C.B. and therefore P.G. would have escaped infection. This illustrates the need for emergency action on the part of the practising physician and of the health official, since G.D. went back to school on April 14. It is instructive to note that had these stricter quarantine measures been employed, the termination of the outbreak might well have been ascribed thereto, since this was the first outbreak of the year and appeared to have been imported. In fact, only two more cases occurred even without any tightening of the usual public health measures.

SUMMARY

1. An outbreak of poliomyelitis in Toronto in April 1955 is described.

2. The value of tissue-culture methods in the epidemiological investigation is emphasized.

3. The outbreak appeared to be introduced by a person infected in Mexico.

4. The Type 1 virus, which was both communicable and invasive, was recovered from all the seven patients and two family contacts.

5. One child attended a day school in the minor illness phase of infection, but only one secondary case developed in the class.

The co-operation of the following Toronto physicians is gratefully acknowledged: Dr. Jean Leeson, Dr. M. J. O'Brien, Dr. G. McNaughton, Dr. W. Jacques, Dr. C. E. Hill and Dr. J. M. Glenroy.

RÉSUMÉ

Les auteurs désirent mettre en évidence le progrès apporté en épidèmiologie par la découverte de Enders, Weller et Robbins de la conservation en tissu de culture des virus de la poliomyélite. Les considérations présentes s'appliquent à l'épidémie qui a sévi à Toronto en avril 1955 et dont la source aurait été un résident de la localité récemment revenu du Mexique.

Le milieu de culture employé dans ces recherches provenait du rein de singe traité à la trypsine. Les virus étaient isolés des matières fécales. Lorsque, à l'examen microscopique quotidien, des altérations cytopathogéniques étaient notées, les tissus étaient soumis au typage grâce à un anti-serum de singe préparé d'après les trois types sérologiques de virus de poliomyélite. Le titrage des anticorps neutralisants était pratiqué d'après la méthode de Salk. Suit une brève description de l'épidémie et de ses sept principaux cas. Le virus incriminé aurait été du type no 1, d'ailleurs responsable de 90% de tous les cas vus au Canada. Il est à remarquer que les 2 malades adultes possédaient un titre de 1:16 contre les trois types de virus, alors que les enfants n'avaient un titre élevé que pour le type no 1. La valeur de la méthode nous est montrée par la confirmation qu'elle apporte au diagnostic clinique de poliomyélite difficile à soutenir au Canada en avril. Deux malades montrèrent un titre sérologique aussi élevé pendant la phase aiguë de la maladie qu'au cours de leur convalescence, ce qui fait ressortir l'avantage de la méthode basée sur l'isolation du virus en comparaison de celle bassée sur la variation des titres d'anticorps. On doit remarquer la rapidité avec laquelle le virus se propagea chez les enfants de deux familles, en contraste avec la contagion très limitée qui eut lieu à l'école. Il semblerait qu'une période de quarantaine de 3 semaines pourrait remplacer avantageusement la période de 7 jours en vigueur dans notre pays. M.R.D.

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WHAT MAY WE EXPECT OF THE AUTOMOBILE SEAT BELT?

TWENTY-TWO MORE CRASHES WITH BELTS

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AT THE OUTSET, let it be clear that no brief is to be made for the seat belt as distinguished from any other restraining device. The seat belt was originally devised to keep airplane pilots in their seats in rough weather, but it has been called upon to do things for which it was not designed. We need a new device, designed to keep the motorcar occupant in his seat.

The important thing for the modern motorist to understand is that his major problem is to avoid blows to his head. Rare is the motorist with an adequate concept of the forces that assail him when his car comes suddenly to a stop. He must come to realize that when the car stops suddenly, he continues forward at the same speed the car had at the moment of impact. As stated by Drs. Marsh and Moore,[5] "The fall out of an airplane will not hurt you, but the sudden stop will."

As pointed out by Harper,[4] when a 200-pound individual comes to a stop from a speed of 30 miles an hour, the force of his impact will vary from one and a half tons up to *eighteen* tons, depending upon whether he comes to a stop in two feet or two inches. Most people can absorb a blow of one and a half tons if the force is fairly well distributed. If the force is distributed evenly over the whole surface of the back, even eighteen tons can be absorbed with survival. But in the usual crash, these gigantic forces are concentrated upon relatively small areas, the face, the head, the chest, with the results that would be expected if the magnitude of these crash forces were understood.

In 1942, Mr. Hugh De Haven[3] published a study of basic significance to this problem. He reported 10 cases of survival in falls from heights up to 150 ft. during which speeds up to more than 60 miles per hour were attained. Survival was achieved: (1) because the force was widely spread over a large area of the body; and (2) because the contacting surfaces allowed from four to 18 inches (average between eight and nine inches) for the absorption of the fall. This brilliant study, and others which have followed it, clearly indicate that planned arrangements can be made whereby the human body can survive falls or crashes which have heretofore been supposed to be necessarily fatal.

The logical way, of course, to prevent traffic injuries is to prevent the "accident", and we must redouble our efforts to do this. The medical profession has yet to comprehend its responsibility in regard to selection and training of drivers. Thousands of persons are driving cars on our streets and roads who should never have been allowed to do this, *and their doctors know it.* Crashes with these people as drivers are no accident, they are inevitable. We must co-operate and even lead in work on the various methods to prevent "accidents."

However, an approach that has been but little explored is the prevention of injury as distinguished from prevention of the accident. It is the considered opinion of this writer that nothing that we can do will produce such a definitive drop in our motorcar death and injury rates as active measures to prevent injuries in spite of accidents.

The magnitude of these crash forces has been indicated, and there are two methods of dealing with them. One is to pad the surfaces that the motorist will strike as he hurtles forward, and the other is to prevent his hurtling forward.

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Actually, both of these methods must be adopted by the carmakers if we are to make any progress in solving our traffic injury problem.

Details of the former include transforming the whole right two-thirds of the instrument panel area into a *crash* panel, a smooth, resilient structure of light metal padded with four inches of one of the newer plastic foams. The car structure must allow a completely unoccupied space at least four inches deep into which this metal panel may be deformed, thus allowing a total of eight inches for the deceleration of the 60-mile-per-hour human projectile. Think of the thousands of dead and hundreds of thousands of mutilated who might have been spared had the motormakers had the good sense to incorporate this structure in their cars since the time, say, when they adopted four-wheel brakes.

At least two inches of this foam material should be placed above the windshield and back along the roof for three or four feet, and as much as can be used, without interfering with proper visibility, on the corner posts. Next, efforts should be multiplied to develop a resilient plastic windshield which will yield as much as eight inches on impact in any weather. Work along this line is in progress. The steering wheel and column should and can be designed to act also as an energy-absorbing device; in fact, the driver has a special advantage in this respect. The levers and knobs that now wreak havoc with motorists' shins and knees, and seem to this writer to have been designed for this purpose, can be placed and designed with a different objective in mind. Mathewson and Severy[6] have pointed out that at least half of the automobiles manufactured will at some time during their useful lives be involved in an accident in which one or more passengers will be thrown into the forward structures. An editorial writer in *The New York Times*[8] suggests, "Thus it would seem incumbent upon automobile manufacturers to view every car coming off the assembly line as though it may be involved in a crash, and to design it with that assumption in mind. Preoccupation with the design of hoods and tail lights should give way to a greater concern with the centre and interior of the car, where, after all, its occupants are." Lastly, and probably most importantly, the carmakers must provide us with doors which stay shut. The present doors pop open at the least impact, even in late model cars, to spill passengers out against curbs, poles, and trees, or into the path of other cars. The motormakers boast that their cars are built to save lives, but a recent study by Cornell's Crash Injury Research group[7] reveals that death and injury are significantly greater if one is involved in an accident in a 1950-54 car than if one is in an "old" 1940-49 car, and it is not because the new cars go faster.

The second approach to our problem is to prevent the motorist's leaving his seat in a crash. No safer place can be devised for him than his seat. For all occupants, except of course the driver, the seat would be safer if it were turned to face the rear, and were solidly anchored to the floor of the car so that it would not tear loose. Military aircraft personnel routinely face the rear with their backs and heads against a bulkhead when making a crash landing, an attitude almost exactly duplicated by rearward seating. Only one further modification in the seat need be made, i.e. the seat back must be extended high enough so that the head is well supported. And there must be a seat belt, also, to hold the passenger in the seat in case the car overturns or spins. The initial severe impact is taken on the back and the belt reverts to its original role, which it performs admirably.

It will be interesting to observe whether we ever adopt rearward seating in the motor car. In aviation, it is becoming more common in certain areas in the United Kingdom, and it is to be hoped that the contagion will spread to this continent. Air transport has come to stay, and we pay for safety whether we get it or not, so it is time for us to face facts squarely and face our seats toward the rear, at once in airplanes and as soon as possible in motorcars.

Until that happy day, a great deal may be done by adopting the seat belt as we now know it. It is not enough; to it should be added at least a single shoulder strap, preferably a double shoulder strap, or a horizontal separate chest belt; and Severy and Mathewson have introduced the idea of a double shoulder loop, possibly to be used without the seat belt. Whatever the device found eventually to be best, some means must be found to keep the motorist in his forward facing seat in the event of a crash, and to date the most accessible device is the seat belt. Whatever the belt may not do, it at least keeps the motorist in the car, and that doubles his chance of survival.

While laboratory studies indicate that the lap belt allows the head to swing forward and strike the forward structures (more reason why these should be padded and constructed as indicated above), most of the laboratory exercises to date are with forces almost strictly parallel to the long axis of the car. These forces obviously will generally be the major component in any crash situation. Nevertheless, on the highway, almost every crash involves powerful lateral or spin forces, and actual highway experience to date indicates that the seat belt is affording a remarkable degree of protection. We urge its immediate installation in all cars now on the road. Further, we urge the addition of shoulder straps or chest belts for the added protection which they quite naturally afford (see Case 29 *infra*).

Some may consider the extensive remodelling of the forward structures unnecessary if adequate restraints are used. This is quite logical, but ignores the human equation. Many people will refuse to use any kind of restraints, preferring to "take their chances". Others will neglect to fasten restraints. One ingenious inventor has suggested a barrier which descends from the roof in case of impact. We must employ *all* methods to prevent accidents and to prevent injuries.

The soundness and importance of these proposed structural changes in the motorcar are indicated by the fact that the American College of Surgeons[9] has sent a formal resolution to the motorcar makers of America recommending "that they stress occupant safety as a basic factor in automobile design, to include (1) doors which will not open on impact; (2) seats and cushions which will not become displaced on impact; (3) energy-absorbing interiors; (4) adequate safety belts or other passenger stabilizing devices that will resist impacts of at least 20 G's."

While resolutions of this type are important, and more organizations must bring such resolutions to bear upon the industry, they are not so important as a "clamour at the box-office." Every doctor, and as many of his patients as he can influence, must demand safety of their dealer, must indicate that they will pay for safety just as they now pay for "flair" and "style" and "colour." We do not need to choose between safety and style; actually, the safe car can be even more attractive than the conventional car of the moment. The objection that these safety changes will cost too much is silly; the lack of them is now costing us astronomical sums in the form of hospital bills and days lost.

Elsewhere[2] we have recorded 19 instances of crashes with belts, with one fatality and one severe injury. In the others, the injuries were minor or nil despite very severe crash conditions in the majority. Since that report some 22 further cases have been brought to the attention of our committee.

In a personal communication, Commissioner Bernard R. Caldwell, of the California Highway Patrol, describes 10 cases dealing exclusively with patrol units driven by traffic officers at the time of involvement in collisions wherein safety belts were a factor. These have been published in a recent issue of Traffic Digest and Review,[1] and will be summarized here briefly. Safety belts have been standard equipment in all new California patrol cars equipped for the road since April 1953, and at present 540 of 740 Highway Patrol cars in operation on California highways are equipped with safety belts.

This first case is not given a number, because, as will be seen, it did not actually involve the use of belts. We have two other cases in which belts, although present in the car, were not fastened. A fatality resulted in each case, and we presume of course that death and possibly injury would have been avoided if the belts had been fastened. These cases are all instructive, but will not be listed as cases of "crashes with belts".

March 13, 1954, 11.25 a.m. Officer was pursuing speeder when violator's vehicle broadsided out of control. Patrol car swerved to miss violator, skidded and broadslid into telephone pole. Impact was such that pole pushed front seat forward 24 inches. The car, a 1953 Oldsmobile Tudor, was totally demolished. Impact hurled officers from car on to soft ground and minor injuries were sustained. Both officers felt that had they been held securely in place by safety belts they might have suffered internal injuries when the seat moved forward and possible head injuries from being whipped against the telephone pole. They considered themselves fortunate that they were not using belts at the time.

The obverse of this situation is provided by the following case.

Salida, Colorado, December 27, 1954. The car skidded sideways over a bank while rounding a curve on Monarch Pass, sailed about 100 feet through the air, bounced on the left front fender, plunged another 50 feet before touching ground again, and finally rolled 150 feet more down the bank before bouncing to a stop on all four wheels. The driver was only slightly hurt, and her mother, age 76, was hospitalized with serious back injuries. She left the hospital 21 days later wearing a cervical spine support.

Had these motorists been wearing seat belts, this writer, for one, would have credited belts with a most remarkable "save".

CASE 20. March 7, 1954, 1.45 a.m. An officer was pursuing a speeder when he entered a congested intersection at a speed of from 55 to 60 miles per hour. Concentrating on intersection traffic, the officer was unaware of a raised railroad crossing immediately beyond. He struck the elevated crossing, the car bounced, and the front end dug into the pavement. The hood flew up and struck his windshield, but the officer, held in position by his safety belt, was able to bring the car to a smooth gradual stop without losing control. Damage to the vehicle, a 1953 Oldsmobile Tudor, was approximately $500. The officer was uninjured.

CASE 21. November 16, 1954, 7.12 p.m. While the patrol car was pursuing a speeder at a speed of from 70 to 75 miles per hour, a slow-moving vehicle in the right-hand lane, in order to pass a truck, moved to the left lane in front of the Highway Patrol car. To avoid colliding with the passing vehicle or the truck, the Patrol car was steered from the roadway down an eight-foot embankment and into a culvert. Injuries to both officers in the vehicle were classified as minor, though one of them suffered three broken bones in his left foot. The Patrol car, a 1954 Oldsmobile Tudor, was damaged to the extent of $757.95. Both officers declared their belief that the safety belts had prevented serious or fatal injuries.

CASE 22. November 27, 1954, 8.00 p.m. A Highway Patrol vehicle was pursuing a violator at a speed between 60 and 65 miles per hour when the vehicle ahead attempted a U-turn. The patrol car struck the right front bumper of the other automobile, went off the roadway and overturned. The officer operating the patrol vehicle was slightly injured, the fellow officer riding as passenger was uninjured. The vehicle, a 1954 Oldsmobile Tudor, was completely demolished.

CASE 23. December 8, 1954, 2.20 p.m. While responding to an emergency call with red light on and siren being operated as required, the Patrol vehicle started to pass a yielding vehicle when another vehicle entered the arterial road from a side street. In dodging the car entering from the side street the Patrol car struck the other vehicle in the left rear. The Patrol car's rate of speed was estimated at 60 miles per hour. The traffic officer suffered torn muscles in his right shoulder. Damage to the Patrol car, a 1953 Oldsmobile Tudor, was $200.

CASE 24. December 8, 1954, 4.25 p.m. Responding to an emergency call at a speed of 70 miles per hour, the officer was suddenly confronted by a vehicle which undertook to turn left in front of the Patrol car. In avoiding the vehicle the officer steered on to the soft shoulder, the Patrol car went out of control, struck a utility pole, overturned, righted itself and came to rest about 300 feet from the point where it had left the roadway. The officer suffered pulled ligaments in his thigh, back, neck, and chest. Vehicle damaged to the extent of $624.32. Officer declared, "Thank God for safety belts."

CASE 25. December 9, 1954, 2.25 p.m. An officer was travelling at a moderate speed of 15 miles per hour on routine patrol on a snowy day. A vehicle approaching from the opposite direction went out of control, crossed the centre of the roadway, and struck the Patrol car on the left front fender. Damage to the Patrol car, a 1953 Chevrolet Tudor, was estimated at $700. The officer received a minor bruise to one hip. It was his opinion that without the safety belt he might have received severe injuries.

CASE 26. March 5, 1955, 2.59 p.m. An officer was pursuing a vehicle at speeds approaching 90 miles per hour, the red light being on and the siren being sounded as necessary. Suddenly a slower moving vehicle changed lanes directly in front of the Patrol car. Swerving to miss the vehicle, the officer applied his brakes, hit the curb dividing opposing lanes of traffic, crossed the opposing traffic lanes, rolled over twice and came to rest some 350 feet beyond the point of origination of skid marks. The officer reported that the use of the safety belt prevented serious injury. The vehicle, a 1954 Oldsmobile Tudor, was considered a total loss.

CASE 27. March 9, 1955, 4.30 p.m. An officer in pursuit of a traffic violator was travelling at approximately 60 to 65 miles per hour when another vehicle attempted to make a left turn directly in front of him. The turning car struck the Patrol car on the left front fender and left door, sending it careening up against an embankment. Damage to the 1954 Oldsmobile Tudor was estimated at between $400 and $500. Three days after the accident the officer wrote: "I wish to thank the members of the administrative staff and other personnel of the California Highway Patrol who are directly responsible for the procurement and installation of safety belts in our regular Patrol equipment. I feel this added safety factor saved me from great bodily injury and possibly saved my life."

CASE 28. March 13, 1955, 8.25 p.m. Pursuing a speeder at approximately 70 miles an hour, the Patrol vehicle suddenly came upon another vehicle starting either a left turn or U-turn from the right shoulder of the roadway. The officer applied the brakes and the Patrol vehicle went into a broad slide, striking the left rear fender of the other vehicle with the left rear of the Patrol car. Following the impact, the officer driving the Patrol vehicle was thrown from the vehicle on to the highway and suffered painful cuts and bruises. The 1954 Oldsmobile Tudor which he was driving was damaged to the extent of $500. The officer has been an advocate of safety belts and had his fastened at the time of the collision. In some manner the buckle became disengaged, allowing the officer to be thrown from the car. A fellow officer speculated that the belt may have offered sufficient protection before failure to prevent fatal or very serious injuries.

CASE 29. Dr. George H. Spielman of Mandan, N.D., was forced to take the ditch to avoid collision with an approaching car which made a left turn in front of him. He and his wife were wearing seat belts of the shoulder and lap type. His car somersaulted into the ditch, landing on its top. Inside, the doctor and his wife sat upside down, suspended by their belts. The windshield was completely shattered. Loosening their seat belts, they crawled out through the car window. This is the second car he has owned with the safety device. They were both unhurt, and agreed, "The seat belts saved our lives."

CASE 30. This case is reported by Mr. Stanley Evans, Head, Investigation Division, Naval Ordnance Plant, Forest Park, Illinois.

"While I was driving to work one morning recently with my wife, a car bumped into my car and caused minor damage to my car's bumper and trunk. I got out of my car and was examining the damage while my wife remained in the front seat strapped with her safety belt. Suddenly a speeding car loomed up along the highway and rammed into my car, shoving it along the road about 100 feet. Despite the terrific impact on my car and the great damage done to it, my wife was completely unharmed. I'm almost certain that she would have been severely if not fatally injured if it had not been for the safety belt which was installed only a few

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days before the accident." Further report reveals, "The car was struck squarely in the rear by another car going from 45 to 50 miles per hour. The rear end of Mr. Evans's car was completely smashed in and the car was eventually classed by his insurance company as a total wreck."

It is the opinion of this writer that the belt in this case really had very little to do with Mrs. Evans's escape from injury, and that actually the case brilliantly illustrates the effectiveness of the rearward seating position in preventing injury. In the absence of a high seat back, it is indeed fortunate that she did not acquire an injury to the cervical spine.

CASES 31 TO 41. Mr. Roscoe A. Stanford, Director, Financial Responsibility Section, Division of Motor Vehicles, Arizona Highway Department, writes as follows: "From January 1954 to May 1955 I noted eleven individuals declaring the use of safety belts who were involved in automobile incidents reported to this agency. My observation at the time of handling each case was to the effect that none of the eleven gave an account of any serious injuries, but all gave property damage in excess of $200. Whether or not the safety belts were actually in use was not indicated on any report. Beginning July 1, 1955, this office will endeavour to obtain accurate information on the use of such safety devices."

We strongly urge all motor vehicle departments to commence recording on the original accident report the presence and type of restraining devices, if any, and whether they were fastened at the time of the crash. In this way, voluminous and accurate information as to the effectiveness of these devices may soon be available.

SUMMARY

Our motorcar deaths and injuries occur because the motorcar manufacturers make no provision for the control of deceleration of the car occupants when the automobile stops suddenly. Design and construction of the car interior with this purpose in mind will change the entire aspect of our motorcar crash problem. These changes will be brought about when the motoring public demands them. For the cars now on the road, the immediate installation of seat belts, preferably with some form of shoulder restraint, will save a large proportion of the lives due to be lost and prevent an even larger proportion of the injuries due to occur without these devices. Many thousands of people are constitutionally incapable of driving motor cars successfully. The medical profession must accept its responsibility in denying the privilege to these people. Forty-one crashes with belts are reviewed.

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ADDENDUM.—Since this paper was submitted for publication, the number of crashes with belts has come to total 87. There were eight fatalities, four in cars in which the occupants had not fastened the belts. The other four fatalities occurred in open cars, two of which overturned. There were three serious injuries, two of which would have been lessened by shoulder straps. Most of the remainder had no injuries at all; a few had relatively mild injuries. Many of the crashes were of the kind labelled by the authorities as surely fatal.

A SYSTEM OF VISION TESTING FOR MOTOR VEHICLE DRIVERS

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AT FIRST GLANCE this subject seems to be relatively simple. But one does not have to consider it very long, or read much of the relevant literature, to become aware of its extreme complexity.

How well one must see in order to drive safely is a difficult matter to decide. The reason of course is that both vision and the driving situation are complex phenomena. The literature which has evolved about these matters is both voluminous and confusing. Often experts of great eminence take diametrically opposed views and, to make the formation of sound opinions more difficult, there is very little adequate experimental work. It seems to be axiomatic of human behaviour that when problems cannot be reduced to simple proof, opinions regarding cause and effect will be legion.

It is possible to find one's way through this confusion of ideas only by holding steadfastly to first principles. The total function of *sight* involves not only *perception*, but also *interpretation*. The difficulty in assessing the relation of sight to driving competency is that *interpretative* action of the intellect may compensate for defects in *perception*. Also the antithesis is true, for even the most refined visual *perception*

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will not be adequate for driving safety if *interpretation* is upset or defective. Thus, we have an experimental situation involving three variables: *perception, interpretation,* and the *driving situation.* To study the relation between visual perception on the one hand and driving ability on the other, the factor of interpretation must be controlled. Such studies are of basic importance if one is to set up rational visual standards for drivers. Unfortunately, controlled studies have not been carried out, because of the extreme difficulty, or even impossibility, of controlling this factor of interpretation.

If for no other reason than this, it is not possible to reduce to simple cause and effect the relation of visual characteristics to driving performance. But this is not the only difficulty. Visual functions cannot be measured exactly. The U.S. Navy[1] in a comprehensive study of visual acuity and heterophoria measurements, using the orthorator, telebinocular and sight screener, found relatively high test-retest reliability for visual acuity and lateral heterophoria only. In a more recent and similar study of school children[2] good reliability for visual acuity tests was found, but heterophoria measurements were less repeatable. These findings confirmed other studies carried out by the R.C.A.F. during World War II. Thus it is not difficult to understand why there are so many opinions in the literature regarding the relationship of specific visual factors to driving safety.

Though a highly scientific approach is impossible, yet by keeping the above basic principles in mind, and using what is known at present, it should be possible to arrive at some reasonable opinion on the manner of testing the vision of motor vehicle drivers.

When setting up visual testing procedures the following matters must be considered:

(a) The visual characteristics to be tested. (b) The level of acceptance and the rejection of candidates. (c) The manner in which the tests are to be carried out.

Even when the visual functions to be tested and the standards of acceptability have been chosen, one must always keep an open mind and preserve a scientific attitude. With the passage of time and the accumulation of correlated data relative to thousands of cases, it should be possible to reduce the variables so as to arrive at an increasingly accurate standard of vision for the average type of person driving under average conditions.

Having decided what factors in visual function are to be tested, and what are to be the standards of acceptance, we must choose test procedures which: (a) have a high test-retest reliability, and (b) are easy to administer quickly by relatively inexperienced people to large numbers of people.

In discussing these matters, the majority of authors consider the following visual factors to be of importance to the motor vehicle driver: (a) visual acuity, (b) fields of vision, (c) ocular muscle balance, (d) stereopsis, (e) colour vision, and (f) night vision.

VISUAL ACUITY

The majority of experts who express an opinion in the literature feel that a certain level of visual acuity should be required of all drivers. European opinion in this matter has been summed up by Coppez,[3] and a comprehensive review of European literature and European standards was made by Jeandelize[4] in 1928. Further, Weekers[5-7] has written extensively. Most of these authors feel that visual acuity should be standardized to the level of about 20/40. At the XIIIth International Congress of Ophthalmology[8] held in Amsterdam in 1929, these European opinions were consolidated to the following standards:

(a) *Public service:* Visual acuity must be 20/20 in one eye and 20/30 in the other. Glasses are not permitted up to 40 years of age. After this, corrective glasses up to 4Δ ametropia are to be permitted. Visual fields (150° in the horizontal meridian in each eye) and colour sense must be normal. Re-examination is to be every three years.

(b) *Taxis:* The same visual acuity standards as for (a) but glasses are allowed from the first and up to 5Δ ametropia is permitted. Visual fields and colour sense must be normal. Re-examination is to be every five years.

(c) *Private cars:* Binocular visual acuity after correction must be 20/60; 20/200 permitted in the worse eye. Visual fields somewhat less than above are allowed (100° in the horizontal meridian). Monocular persons are accepted if the acuity is 20/40 and the visual field is normal, but the person must have had at least one year to become used to the one-eyed condition. Persons with visual acuity below 20/200, or with non-admissible visual fields, should be considered monocular. Periodic examinations are desirable.

Seven of the 10 provinces in Canada require visual acuity tests, the permissible level ranging from 20/40 to 20/70. In 46 of the 48 States of the United States, visual acuity standards ranging from 20/30 to 20/70 are required.

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There is very little experimental basis for these standards, however. Fletcher,[9, 10] studying the relationship of visual acuity to accident frequency, found that in "non-repeaters" the visual acuity generally was superior to that in "repeaters", but this observation was not supported by the findings of De Silva, Robinson and Forbes,[11] and of Brody.[12] In an excellent study by the Eno Foundation[13] little evidence was found that "repeaters" have poorer visual acuity than "non-repeaters". Brody[14] in a recent paper feels that visual acuity has been over-emphasized.

I believe that visual acuity measurements are one of the few places in this problem where we are on relatively firm ground. In the R.C.A.F. in 1946 McCulloch and Crush[24] showed that there is a definite relation between visual acuity and depth perception. Other R.C.A.F. studies[15] showed that there is a definite correlation between visual acuity and ability to land an aircraft safely.

Thus there is a certain amount of experimental evidence, and there certainly is a weight of expert thought supporting the opinion that a visual acuity standard is required. The U.S. Navy[1] and schoolchildren[2] studies mentioned above indicate that visual acuity is one of the few visual factors which can be reliably measured. From the medical point of view, a visual acuity test is the starting point in the examination for all ocular diseases and thus for this reason alone it should be included.

VISUAL FIELDS

Brody[12] found that a defective lateral field of vision was a significant factor among "repeaters". In a recent paper[14] he enlarges upon this idea. However, it must be pointed out that the Eno study[13] could find no relationship between this defect and accident occurrence. Berens[16] feels that a field of vision less than 104° tends to make for unsafe driving. Thus, he substantially agrees with the recommendations of the XIIIth International Congress of Ophthalmology.[8] For this reason and because the visual field is often affected in ocular disease, I feel that a peripheral field test, in the horizontal meridians at least, should be carried out. This is relatively accurate and reliable. Faulty response to this test should be a cause for referring the candidate to a competent ophthalmologist.

OCULAR MUSCLE BALANCE

Results of various studies relating heterophoria and heterotropia to safe driving are equivocal. This is in keeping with the findings of the R.C.A.F.[15] during World War II which showed that in general poor depth perception cannot be related to variations in ocular coordination. Many other studies support this view. On the other hand, Lauer and Algaier[16] report that an inability to use the eyes together equally and simultaneously is a factor among "repeaters". This finding was supported by those of the Eno study.[13] Weiss and Lauer[18] also found "repeaters" to be deficient in ocular muscle balance.

Added to this lack of consistent evidence that heterophoria or heterotropia has any relationship to the ability to drive a car safely is the fact that there are no simply applied tests of high reliability for these factors.[1, 2] Thus, I believe that for the present time this measurement should be left out of the visual examination. Persons complaining of diplopia should be referred for eye examination by a competent ophthalmologist. This is also the recommendation of continental experts (Jeandelize[4]).

STEREOPSIS

When one comes to the matter of stereoscopic vision, the pinnacle of binocular function, the various studies relating it to safe driving are even more contradictory. Fletcher[9, 10] as well as De Silva, Robinson and Forbes[11] found poor stereopsis to be a factor associated with accidents. In one part of the Eno study[13] this was confirmed, but in another part it could not be substantiated.

The various tests for stereopsis are even more unreliable than those for heterophoria. The R.C.A.F. in World War II carried out a comprehensive survey of all known tests for binocular depth perception, or stereopsis. It was found that the Howard Dolman type of test was entirely unreliable.[17] This was true also of various types of stereoscopic slides in the major amblyoscope. None of these was reliable and none correlated with variations in ocular muscle balance, or with the ability to land aircraft safely.

The only reliable test for stereopsis found was a modification by Kirschberg of one developed by Verehoff.[19] This test was found to be highly reliable and repeatable. However, the refinement

of stereopsis, as measured by this instrument, could not be correlated with variations in ocular muscle balance or with the ability to land an aircraft. Thus, it appears that not only is stereopsis a difficult matter to study and accurately evaluate, but also it is likely that it has little if any relation to the ability to drive a car safely. I recommend that for the present all such tests be left out of the examination.

COLOUR VISION

Lauer[20] has demonstrated that colour blindness has no significant relation to the frequency of accidents. This finding has been fully authenticated by other studies in the U.S.A. and by studies in Europe. Selling[21] has pointed out a number of good reasons why this should be so.

NIGHT VISION

The Eno study[13] indicated that there is no correlation between dark adaptation and the incidence of accidents. This is the only extensive study on this topic. It made use of the Feldman adaptometer, which is recognized to be an inexact instrument. Also, motor driving at night with our modern powerful headlights does not call upon rod vision. Thus, the lack of correlation found between night blindness and accident rate is not unexpected. Further, night vision is an extremely difficult function to test accurately. It was the experience of the R.C.A.F. in World War II that the only way in which it can be measured adequately is by a battery of tests involving much time. These tests make use of several expensive and highly technical instruments; therefore testing this function at the present time is far outside the capacity of any automobile licensing bureau, and takes too much time to be feasible.

Of course the importance of being able to see in the dark must not be underestimated, since the number of accidents at night is several times greater than that during daytime. As Davson[22] has so neatly expressed it; if the visual acuity in daylight is taken as 100, the visual acuity in full moonlight is only 35, in half moonlight 16, in clear starlight 8, and under an overcast sky only 4. More important is the fact that this function rapidly deteriorates after the age of 45 years. This deterioration is supposed to be due to senile changes in the circulation of the choroid and retina.

Even though accurate tests for night vision cannot be carried out easily, one may reach the same end by referring all patients with defective visual acuity and/or fields to an ophthalmologist. In this manner pathological conditions leading to night blindness will be discovered. When one comes to the elderly driver, it is a question whether he or she should be permitted to drive at all.

TABLE I.

RECOMMENDED VISUAL REQUIREMENTS

Type of licence	*Corrected visual acuity*		*Refractive error (eq. power)*	*Fields (horizontal)*	*Re-exam.*
(1) Drivers of heavy transport.....	20/30	20/30	Not more than 4 ⊿	Full (140°) each eye	Every 3 years
(2) Drivers of light transport.....	20/30	20/60	Not more than 5 ⊿	Full (140°) each eye	Every 3 years
(3) Private drivers.					
(a) Unconditional.....	20/40	20/100	Not applicable	Full (140°) in one eye; not less than than 100° in other	Every 3 years
(b) Conditional. Monocular, strabismus and cataract candidates licensed on their merits.					

RECOMMENDED VISUAL EXAMINATION

Following the above discussion and keeping in mind the principles there outlined, it should now be possible to arrive at a reasonable system of visual testing for automobile drivers. At the present time the only defensible type of examination is one which involves no more than tests for visual acuity, and visual fields in the horizontal meridian. In those cases where it seems to be necessary, this should be backed by an eye examination carried out by an ophthalmologist. At licensing bureaus I believe that one could do no better than follow the basic pattern for visual testing laid down by the XIIIth International Congress of Ophthalmology, with certain modifications and refinements owing to the enlargement and clarification of knowledge in the subsequent 25 years. I like very much the procedure outlined there, whereby different levels of vision are required depending on the difficulty and responsibility of the driving situa-

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tion. The Congress made the requirement for visual acuity rather stricter for certain categories than is thought necessary nowadays. Certainly this is true among the best workers on this continent. Also, it is now felt that colour vision testing is not necessary, and the testing of the whole visual field is over-exacting. Save for these modifications then, the following system of vision testing is recommended (cf. Table I).

Drivers of heavy transport and public vehicles.—Visual acuity must be at least 20/30 in each eye, either with glasses or without. Where glasses are required, the equivalent power of the refractive error must not be greater than 4Δ in either eye. Where vision is not fully correctible, the applicant should be referred to an ophthalmologist for examination. The ophthalmologist may make recommendations, but the final decision on acceptance must rest with the licensing authorities.

The fields of vision must be normal in each eye, and must reach to at least 50° on the nasal side, and 90° on the left temporal side, using a 3 mm. white object at 330 mm. under constant illumination of 20 foot candles.

The candidate should be given an eye examination every three years. Where glasses are worn, an ophthalmologist's examination should be required. No candidate with progressive eye disease affecting vision may be accepted in this group.

Drivers of taxis and light transports.—The visual acuity must be at least 20/30 for both eyes, either by means of glasses or not, but the applicant may be permitted to have one eye worse than the other. In this case the error must be correctible to 20/60. Where glasses are worn, the equivalent power of the refractive error must not be greater than 5Δ. The candidate must have full visual fields in each eye, in the horizontal meridians as outlined above.

The applicant should have an eye examination every three years, and an ophthalmic examination where glasses are worn and wherever else seems necessary, such as when the vision is not fully correctible. No person with progressive eye disease affecting vision may be permitted this type of licence.

PRIVATE DRIVERS

(a) *Unconditional licence*

The candidate should have at least 20/40 vision with both eyes, and the vision in one eye should be not less than 20/100 when corrected with or without glasses.

The applicant may be permitted a visual field in one eye reduced to 30° on the nasal side and 70° on the temporal side, provided the field in the other eye is 50° on the nasal side and 90° on the temporal side by the test outlined above. Drivers should have a visual examination every three years. All those whose visual acuity is not fully correctible and those with progressive eye disease affecting vision should be referred to an ophthalmologist and examined at repeated intervals as recommended by him. The ophthalmologist may make recommendations, but acceptance must rest with the licensing authority.

(b) *Conditional licence*

Candidates who are blind in one eye, have a strabismus, or have had a cataract extracted deserve special attention. Each case should be considered on its merits by an experienced ophthalmologist, as so many variations of the basic condition may occur. As a guide, and only as a guide, the following opinions regarding certain specific cases may be expressed. This discussion may serve to emphasize the many problems and form the basis for the collection of data.

Candidates blind in one eye may be permitted to drive provided the vision in the remaining eye is 20/30 or better, with or without glasses. The refractive error in this eye must not be greater than an equivalent power of 5Δ. The field of vision in this eye must be normal; that is, to 50° on the nasal side and 90° on the temporal side, using the test outlined above. Applicants with a visual acuity of 20/200 or less when corrected, and those with a field smaller than 30° on the nasal side and 70° on the temporal side, are to be considered one-eyed. Such one-eyed persons may be permitted to drive a car at speeds less than 40 miles an hour on the open road and 25 miles per hour in the city, provided that they have had six months to become accustomed to their monocular condition, and that there is evidence indicating a high order of moral character and intelligence.

Candidates with strabismus require some special consideration. If they totally suppress vision in one eye, they should be treated as though they were monocular and as outlined above. But if they merely suppress the central area of the field and maintain a full peripheral field, then I believe it is quite safe to permit

them to drive, provided the corrected vision of the fixing eye is 20/30 or better.

Candidates who have had a single or bilateral cataract extraction present a problem. If one cataract has been removed and the vision in this eye when corrected is a full normal, and if the vision in the other eye is at least 20/30 when corrected and there is no evidence of a cataract or any other disease in it, then I believe the candidate should be permitted to drive, but under the conditions as outlined above for monocular individuals. If, however, the vision in the unoperated eye is less than 20/30, or if a cataract is present in it, then I believe he should not be permitted to drive at all. People who have had a cataract extracted from each eye may be permitted to drive with the limitations of a monocular person if the corrected vision is 20/30 in each eye, if there are full fields and there is no diplopia with glasses on.

It will be recognized from the above that there are many types of cases which require very special consideration. A scientifically justifiable and morally equitable decision can be made in these cases only by a properly trained, highly experienced ophthalmologist. At this point I cannot recommend too strongly that the licensing authority appoint such an ophthalmologist or group of ophthalmologists to adjudicate such cases. Such an authority should have administrative powers to see that properly trained technicians are available to carry out the preliminary examination of candidates, and to see that proper equipment is obtained and that it is kept in order. Also it should organize and supervise a research programme.

METHODS OF EXAMINATION

It is manifestly impossible in a paper such as this to discuss critically all the various means of testing all the visual characteristics. However, it is possible to indicate the procedures recognized as best at the present time.

In this province, when vision testing was instituted about two years ago, the equipment and test procedures elaborated by the American Automobile Association were adopted. A committee of the Montreal Ophthalmological Society reviewed these critically about 18 months ago.[23] The tests include one for visual acuity (a form of Snellen's test chart), one for depth perception using a modification of the Howard Dolman procedure, perimetry in the horizontal meridians and a test of night vision.

The committee of the Montreal Ophthalmological Society felt that the visual acuity test was adequate though it was applied in an unnecessarily complicated manner. As for the test of depth perception, basing their opinion upon the extensive studies of the R.C.A.F. during World War II[15, 17, 23] they felt that it was so unreliable and so poorly correlated with actual depth perception in the field that it had no basis for acceptance. It was felt that the test for field of vision was adequate; and that the test for night vision was unacceptable both because of the faulty instrument used and the poor test procedure.

Recently, at one or two of the testing bureaus in the Province of Quebec the Bausch and Lomb orthorator has been introduced. It has also been used in several parts of the U.S.A. Hence, a word should be said here regarding the instruments designed for comprehensive visual screening, such as the orthorator, the sight screener and the telebinocular. These instruments have been investigated in detail as mentioned above by the U.S. Navy[1] and by the U.S. Department of Health, Education, and Welfare.[2] These studies show that they are valuable within rather distinct limits and that nothing can take the place of a careful eye examination by a well-trained ophthalmologist. The reliability of these instruments increases with the intelligence of the individual tested. Thus, they have higher reliability in adults than in children. In the latter, they are relatively unreliable except in the matter of visual acuity. Since in the screening of motor vehicle drivers all types of persons must be dealt with, this is a very serious defect. Also, since present knowledge and experience indicate that only visual acuity and fields of vision are of significance in the primary testing procedure, these instruments are unnecessarily complex and elaborate for the purpose.

It is recommended that Snellen's test be used for testing visual acuity. A variety of charts must be used at a carefully controlled working distance of 20 feet, and they must be kept clean and adequately illuminated. An outstanding instrument in this regard and the one accepted by most experts is that developed by the R.C.N. The U.S. Navy also has a similar one of first-class design. Should it be difficult to obtain one of these, the projection type of instrument, such

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as the project-o-chart, will be found equally good for all practical purposes, though theoretically it is inferior. A wide variety of test charts should be used so as to lessen the chance of memorization. When scoring the test the candidate should be permitted to make up to one-third of reasonable errors on any line and yet be given that line as his visual acuity. In other words, if there are nine letters on the 20/20 line he may be permitted three reasonable errors on that line. For instance, he may be permitted to call an "E" a "B", or an "F" a "P".

The perimeter as outlined by the American Automobile Association is adequate, but it would be better if there were some means of controlling the illumination. Also, the background of the test area should be in neutral grey as in the Ferree Rand perimeter. The test object should be a circular white one 3 mm. in diameter. The same conditions may be obtained with several well-known stock perimeters on the market, such as the Ferree Rand and the Aimark projection perimeter.

In conclusion, it must be emphasized that these standards and recommended test procedures are only a starting point. With the passage of time, data must be collected to form the basis for statistical studies whereby the visual requirements may be enlarged or contracted, to fit ever more accurately the needs of the situation. It is suggested that these data be collected in the following manner. For statistical analysis the use of a Holerith machine is a great help. On the cards used in such a machine, it is recommended that the visual acuity with and without glasses and the state of the visual fields be recorded. As each accident occurs, the persons involved should have their cards punched with data relevant to the accident, such as visibility, whether glasses were worn, the state of the weather, the condition of the road, the person apparently primarily at fault, and the time of day. All the possible factors which may have entered into the situation should be recorded. At the end of a period when many thousands of cases have been recorded, the results should be statistically analyzed. It goes without saying that the data collected are no more accurate than the instruments used and the competency of the examiners. Only highly trained, reliable technicians should be used for collection of these data, and the instruments should be of the type described above.

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THERAPY IN INTERMITTENT CLAUDICATION

Experimental and clinical trials were made of a new vasodilator Arlidin (1-(p-hydroxyphenyl)-2-(1'-methyl-3'-phenyl propylamino) propanol), an analogue of adrenaline and ephedrine. Experimentally Arlidin was shown to be of low toxicity and to increase coronary flow in perfused guinea pig hearts. It also produced anæsthesia on instillation into the rabbit's eye and caused marked vasodilatation of the blood vessels of the ear on intradermal injection. A controlled trial with Arlidin was made in 24 diabetic patients complaining of intermittent claudication or muscle cramps at night. Where a placebo helped the condition the patient was dropped from the finally evaluated group. Treatment continued for 3-21 months and disappearance of night cramps or a threefold increase in the distance the patient could walk was considered a good response. The daily dose was 3-12 mg. three times a day. No severe side-effects were encountered. In two cases response to both Arlidin and the placebo was poor; in three cases both measures produced improvement; 19 patients improved on Arlidin alone.—J. Pomeranze *et al.*: *Angiology*, 6: 271, 1955.

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THE TREATMENT OF URINARY TRACT INFECTIONS*

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THE TREATMENT of urinary tract infections remains a problem in spite of the many chemotherapeutic and antibiotic drugs at present available.[1] This is due largely to the multiplicity of infecting organisms with their varying sensitivity to available drugs, to mechanical factors which may serve to perpetuate infection through irritation or interfere with drainage, and to combinations of these circumstances. Unsatisfactory results may often be attributed to failure to give full consideration to these various factors.

It has been repeatedly emphasized and almost as frequently forgotten that bacilluria and pyuria are signs and not a disease. The urine as secreted is a sterile solution and becomes inoculated with pus cells and organisms during its contact with infected tissues. The density of pus cells reflects to a degree the extent of and nature of the lesion, and the organisms reflect the nature of the infection. The number of organisms may be evidence of the severity of the infection, but also it must be remembered that urine can be a culture medium in which organisms may multiply rapidly.

In dealing with urinary tract infection, the situation is no different from that involved in treating infections elsewhere in the body, such as pneumonia, otitis media, or meningitis. The notable advantage in the urinary tract is that drainage is often more easily obtained.

These infections are in the tissues, and in order to destroy the invading organisms it is necessary to deliver to the site of infection adequate concentrations of antibiotic and/or chemotherapeutic agents to which the organisms are sensitive.

This feature is well illustrated in Figs. 1 and 2, which are sections of kidney and prostate respectively demonstrating the deep localization of infection. In cystitis, although the infection does not extend beyond the submucosa, it must be obvious that little may be expected from merely bathing such tissue with a bacteriostatic or bactericidal solution unless penetration into the deeper epithelial layers can be obtained.

In view of these facts it is not surprising that drugs which fail to provide therapeutic concentrations in blood and tissue should fail to effect cures even though they may sharply reduce the number of organisms found in the urine. Control of infection by altering the pH of the urine with diet and acidifying and alkalinizing agents has been largely abandoned. The lethal pH for effectiveness against the various strains of coliform organisms lies either at one or the other end of the scale and is difficult to obtain in any event. Our experience has convinced us, contrary to that of some others,[2, 3] that the so-called soluble sulphonamides, which are absorbed and excreted rapidly into the urine, are not effective unless administered in doses large enough to provide therapeutic blood levels. It has been shown[4-9] that by combining three sulphonamides it is possible to obtain full therapeutic blood and tissue levels with moderate doses, with practically no danger of kidney damage from crystalluria and with considerably reduced incidence of side-reactions. The antibacterial spectrum of the combination is fairly broad and includes the majority of organisms found in urinary tract infections.[10, 11] The antibiotics have proved of value in the management of many infections, and penicillin is still pre-eminently the one of choice for Gram-positive organisms from the point of view of effectiveness and freedom from untoward effects, more especially when administered by the oral route. It remains an efficient agent in combating coccal infections. The complications following the use of the broad-spectrum antibiotics may be serious. Troublesome and prolonged diarrhœa, due to change in the bacterial flora of the intestinal tract, remains a constant threat. Of great practical importance has been the demonstration by a number of investigators[11-17] of a synergistic action when penicillin is combined with sulphonamides against Gram-positive organisms, both *in vivo* and *in vitro*. More recently El Borolossy and Buttle[18] have demonstrated similar effects in infections with Gram-negative organisms.

The present study was made in order to compare the effectiveness and safety in a variety of urinary tract infections of the oral administration of a triple sulphonamide mixture with the same mixture combined with penicillin. The triple sulphonamide tablet contained a total of

*This work was performed while the author was Resident in Urology, Department of Urology, Royal Victoria Hospital.

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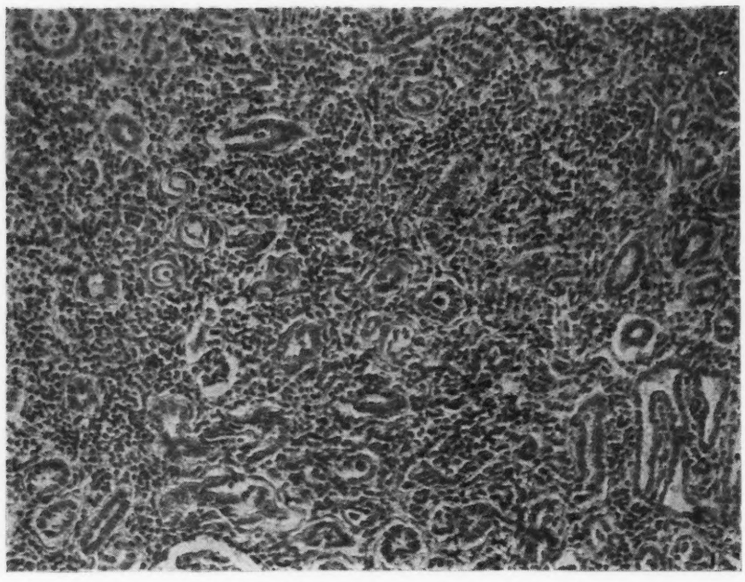

Fig. 1.—Kidney. This section demonstrates the inflammatory involvement of the kidney parenchyma in pyelonephritis.

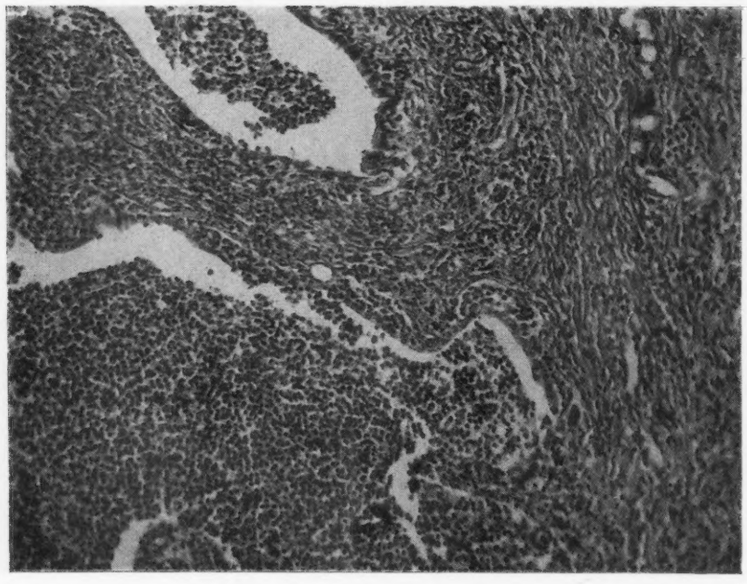

Fig. 2.—Prostate. This section demonstrates suppurative involvement of the parenchyma of the prostate gland.

0.5 g. (7½ grains),* consisting of equal parts of sulphadiazine, sulphamerazine and sulphamethazine, and the sulphonamide-penicillin tablet contained the same amount of sulphonamide plus 100,000 units potassium penicillin G.* The dose was generally two tablets three times daily.

Sixty patients were treated. These had 20 different urological conditions—12 had upper tract disease, 48 lower tract disease. Table I shows the distribution.

TABLE I.

Condition	*No. of cases*	*Condition*	*No. of cases*
Benign prostatic hypertrophy (all underwent prostatic surgery)	20	Carcinoma of the prostate with cystitis	2
Carcinoma of the bladder	6	Balanoposthitis	1
Ureteral calculus	6	Chronic prostatitis	1
Urethral stricture	5	Orchitis (suppurative)	1
Cystitis (acute hæmorrhagic)	5	Pyelonephritis (chronic)	1
Cystitis (chronic)	4	Hydronephrosis (infected)	1
Neurogenic bladder	4	Polycystic disease with pyelonephritis	1
Epididymitis (acute)	3	Renal calculus with pyelonephritis	1
Vesical calculus	3	Infected hydrocele	1
Acute prostatitis	3		
Acute pyelonephritis	2		

BACTERIOLOGY

The predominant organisms encountered, as determined by culture, are listed in Table II.

TABLE II.

Infecting organism	*%*
E. coli	22
*Proteus vulgaris**	17
Str. fæcalis	17
Aerobacter aerogenes	17
Staphylococcus pyogenes	17
Pseudomonas aeruginosa	10

*Without exception the proteus cultures proved to be urea splitters.

Duration of therapy varied considerably and depended on the time required for the relief of symptoms, such as burning, dysuria, frequency, pain and clearing of the urine of pus cells and organisms. It would appear upon analysis of the data in Table III that considerably less time was required to produce such improvement when the sulphonamide-penicillin preparation was employed.

SIDE-REACTIONS

In 60 patients treated, only one side-reaction was noted and this was not of a serious nature. It consisted of nausea and troublesome headache 24 hours after the administration of the combined triple sulphonamide-penicillin preparation.

The following are illustrative case reports:

CASE 1. A.T., male, aged 17, had been under treatment for acute frontal sinusitis and meningitis, with two broad-spectrum antibiotics. He was seen on the Urology Service when he developed acute epididymitis; temperature was 104.5° F.; epididymis swollen and markedly tender. The urine contained an occasional pus cell and on culture grew *Pseudomonas aeruginosa, Staphylococcus pyogenes,* and *Streptococcus fæcalis.* The patient was placed on triple sulphonamide and oral penicillin therapy, as previously described. He became afebrile in 48 hours with satisfactory progress, including the sinusitis, meningitis and epididymitis. This patient appeared to respond promptly and dramatically to combined triple sulphonamide and penicillin therapy in the face of an acute infective process. The duration of therapy was seven days, at which time the urine contained a rare pus cell only.

TABLE III.

Duration of therapy required to produce cure or remission	*Preparation used — Triple sulphonamide with crystalline potassium penicillin G*	*Preparation used — Triple sulphonamide preparation*
	No. of cases	*No. of cases*
1 to 7 days	22	3
8 to 14 days	7	10
15 to 21 days	1	13
22 to 28 days	3	1

CASE 2. H.Q., female, aged 25, was admitted with a five-day history of bilateral loin pain and dysuria, and a temperature of 101.5° F. on admission. There was acute bilateral loin tenderness with splinting present. Catheter urine contained pus cells ++. After investigation the diagnosis of bilateral pyelonephritis was made. Treatment consisted of absolute bed rest, adequate hydration, and administration of the triple sulphonamide preparation alone. The marked pain, the splinting and the fever all subsided in 48 hours. Therapy was continued for 15 days. Upon discharge the catheter urine showed an occasional white cell. There were no symptoms at this time.

*Trulfa-Zine and Trulfacillin manufactured by Messrs. Charles E. Frosst & Co., Montreal.

SUMMARY

1. It has been emphasized that in the treatment of urinary tract infections effective therapy can be achieved only by delivering to the site of infection therapeutic concentrations of antibiotic and chemotherapeutic agents.

2. Sixty patients with a variety of urological conditions were treated with either a triple sulphonamide preparation or a triple sulphonamide preparation combined with penicillin, both of which are known to produce, in adequate dosage, therapeutic blood and tissue levels by oral administration.

3. Good response to treatment was obtained with both preparations. However, duration of treatment was considerably shortened with combined chemotherapy and antibiotic therapy. This lends support to the view that sulphonamides and penicillin act additively and possibly synergistically in the control of infection.

4. Infections due to *Proteus vulgaris* and to *Pseudomonas aeruginosa* remain, in great part, resistant to the chemotherapeutic and antibiotic drugs at present available.

5. The triple sulphonamide preparation or the triple sulphonamide combined with penicillin appears to be a valuable adjuvant in lessening postoperative morbidity after prostatic surgery.

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RÉSUMÉ

On a insisté sur le fait que, pour combattre les infections des voies urinaires, un traitement efficace ne peut réussir qu'en appliquant à l'endroit infecté des agents antibiotiques et chimiothérapeutiques en concentrations thérapeutiques adéquates.

Soixante malades souffrant de troubles urologiques divers furent traités soit par une triple préparation de sulfamidés, soit par une triple préparation de sulfamidés combinée avec de la pénicilline. On sait qu'en doses convenables administrées oralement ces deux médicaments produisent des niveaux thérapeutiques du sang et des tissus.

Le traitement fut couronné de succès avec chacune des deux préparations. Cependant, la durée du traitement fut grandement réduite par l'emploi combiné de la chimiothérapie et des antibiotiques. Ce fait confirme l'opinion que l'action des sulfamidés et celle de la pénicilline se superposent et s'associent peut-être dans la luttre contre l'infection.

Les infections dues au Proteus vulgaris et au bacille pyocyanique résistent, pour la plupart, aux médicaments chimiothérapeutiques et antibiotiques actuels.

La triple préparation de sulfamidés, ou la même combinée avec la pénicilline, paraissent être de précieux adjuvants pour diminuer la morbidité post-opératoire des interventions chirurgicales intéressant la prostate.

M.R.D.

PNEUMATOCELE DISEASE*

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DESPITE A COMMON TENDENCY to minimize or ignore them, abnormal spaces in the lung parenchyma are often just as significant from the point of view of interference with function as are solid lesions. Frequently the defects are more disabling than the masses. Many of the latter, such as hamartomas or fibrous scars, are only of clinical importance to the extent that they must be differentiated from malignant neoplasms. Certain simple defects, however, are liable to complications so serious as to threaten life as surely as any cancer.

Abnormal spaces in the lung parenchyma are somewhat difficult to classify, and have been the subject of much loose thinking and writing. The terms "lung cyst", "bulla", and "pneumatocele" are often used interchangeably with resulting confusion.

While it may be objected that any classification of the subject is open to dispute, it is nevertheless true that a working classification, even if not watertight, simplifies study of the problem, and indeed is essential in order to make the subject clear.

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In that sense, the following classification has proven useful:

1. Infective spaces: (a) Tuberculous cavity. (b) Lung abscess.

2. Non-infective spaces: (a) Lung cyst. (b) Pneumatocele. (c) Honeycomb lung.

3. Neoplastic spaces: (a) Degenerated bronchogenic carcinoma. (b) Degenerated secondary neoplasm.

4. Parasitic spaces: (a) Echinococcus cyst.

This essay concerns itself only with the non-infective defects (group 2 above), and specifically with what we have chosen to call the pneumatoceles. The following definitions of these spaces are acceptable.

(a) *Lung cyst.*—An abnormal space with an epithelial lining, occurring usually as a congenital and rarely as an acquired cavity in the lung. It usually contains fluid and gas.

(b) *Pneumatocele.*—An abnormal space without an epithelial lining, occurring as an acquired cavity in the lung. It contains gas and usually no fluid.

(c) *Honeycomb lung.*—A rare condition, in which a cross-section of the lung resembles a honeycomb or sponge, because of numerous cavities uniformly distributed throughout both lungs. These cavities measure up to about a centimetre in diameter. The pathogenesis of this condition is obscure, but it is known to be associated with xanthomatosis, tubero-sclerosis and pulmonary granulomatosis, and may occur as a result of diffuse bronchiolitis or developmentally.

MORBID ANATOMY

Lung Cyst:

(I) *Congenital*

(a) *Bronchogenic:* (1) Single. This is the so-called sequestrated lung, a cystic structure lined with columnar ciliated epithelium in the body of the lung, but often having its own arterial supply from the aorta.

(2) Multiple. This is seen in children, behaves like bronchiectasis, and is usually called cystic bronchiectasis.

(b) *Alveolar:* Usually seen in children, as a single sizable defect, liable to spontaneous disappearance, and often erroneously called bullous emphysema.

(c) *Mixed: Rare*

(II) *Acquired*

This may occur as the result of a process of epithelization of a burnt-out lung abscess or tuberculous cavity. It must be an extremely rare development. Although its existence is denied by many, there appears to be some evidence that it does occur.

A bronchial communication may or may not be demonstrable with the lung cyst.

Pneumatocele:

(a) Subpleural bleb. A small, bubble-like structure 1-2 cm. in diameter on the surface of the lung, suggesting a gas-containing elevation of the visceral pleura from the lung.

(b) Emphysematous bulla. A much larger and possibly giant-sized, spherical or oval space extending into the lung parenchyma. The fully developed large bulla has the visceral pleura forming a large part of its wall, but smaller bullæ may be buried deep in the body of the lung.

Both of these structures may be single but are usually multiple, and there is a possibility that a subpleural bleb may develop into an emphysematous bulla. A bronchial communication may be only intermittently patent, but is always present. These structures appear to be acquired rather than congenital. Their origin is obscure, but the fact that they are found almost invariably in persons suffering from chronic cough suggests that long-continued forcible cough may have a disruptive effect on the lung structure leading to their causation.

MORBID PHYSIOLOGY OF PNEUMATOCELES

1. *Inflation.*—Owing to the small calibre of their bronchial ostia, these spaces are prone to trap air and thus become inflated to varying degrees. This may lead to pressure effects on neighbouring structures, such as the heart, great vessels and lung, with corresponding embarrassment of function. An even more serious effect of inflation may be its disruptive effect on the surrounding lung parenchyma. Forcible breathing and particularly forcible coughing encourage inflation. Blades[1] points out that at an early phase in cough there is a deep inspiration pulling air into the abnormal space; there follows a period of markedly raised intrapulmonary pressure, when the abdominal muscles contract while the glottis remains closed; next occurs a sudden drop in the intrapulmonary pressure as

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the glottis is suddenly opened, and the expulsive blast of air rushes out; because of the small bronchial communications the air cannot leave the abnormal space as rapidly as it does the normal surrounding lung; consequently, the continued raised pressure in the abnormal space meets little resistance from the lowered pressure in the surrounding lung, which is torn apart as a result.

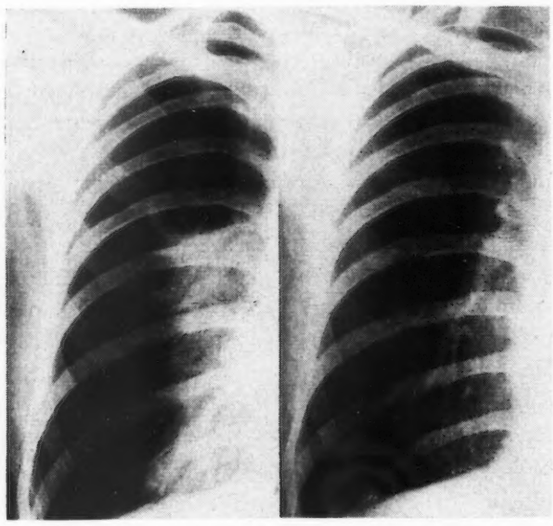

Fig. 1.—Preoperative (left) and postoperative (right) radiographs of case of single pneumatocele complicated by pneumothorax. This suggested a subpleural bleb which had begun to develop into an emphysematous bulla. Its wall was partly destroyed with the cautery.

If this mechanism is active, it may cause subpleural blebs to develop into pulmonary bullæ, and the bullæ in turn may continue to enlarge at the expense of the surrounding lung. This postulates a serious progressive tendency for this disease, and suggests the need for stringent control of chronic forcible cough as well as the urgency of detecting and dealing surgically with bullæ as they arise.

2. *Rupture.*—One of the commonest complications of the pneumatocele is rupture with resulting so-called spontaneous pneumothorax. If the rupture occurs in a subpleural bleb, the pneumothorax may be limited in extent and the lung readily re-expands. The emphysematous bulla, however, has larger and more numerous bronchial ostia, and when it ruptures the pneumothorax may be large, of the distressing high-pressure type, and more liable to persist. This is the type in which repeated air aspiration may prove unsuccessful in bringing about re-expansion of the lung, and surgical treatment is called for.

3. *Increased dead space.*—The upper respiratory passages and the bronchial channels, commonly referred to as dead space because of their relative inactivity in gas exchange as compared with the alveoli, are increased to the extent of the pneumatocele. This interferes in like degree with the vital function of respiration.

4. *Infection.*—It is remarkable, considering their size at times and their apparent accessibility to the bronchial flora, how infrequently the pneumatoceles become infected. The writer has no personal knowledge of and has seen no reports of such infection occurring.

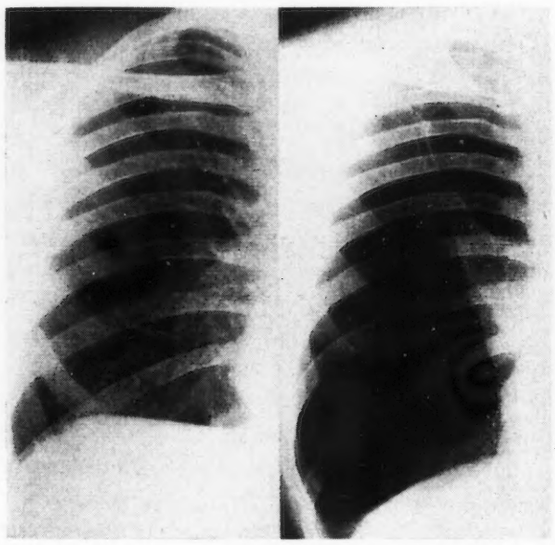

Fig. 2.—Preoperative (right) and postoperative (left) radiographs of case of multiple emphysematous bullæ of the right upper lobe treated by thoracotomy and excision of the bullæ. Thoracoscopy showed the bullæ and proved that what appeared to be adhesions on the radiograph of the pneumothorax were, in fact, the walls of a large bulla which had ruptured.

CLINICAL FEATURES OF PNEUMATOCELES

The patient with pneumatoceles is typically a flat-chested male of a nervous disposition, who oversmokes, suffers from chronic bronchitis, and indulges in periods of forcible uncontrolled cough each day, particularly in the morning. If the condition is advanced, he is dyspnœic on mild exertion, and may be cyanotic even at rest. He may be subject to asthma-like attacks, and in these episodes of bronchiolar spasm there is often an accompanying rise of arterial blood pressure. Periods of acute bronchitis or pneumonitis may increase the distress. Carbon dioxide narcosis is a real danger to these people, and when it becomes necessary to give them oxygen, it should be given sparingly at not more than

three or four litres per minute and for interrupted periods under supervision.

In the early stages of the disease, the alarming symptoms described above will be absent; aside from cough, unhappily regarded by so many doctors and patients alike as a more or less normal phenomenon, no warning of the condition may be given until a spontaneous pneumothorax occurs. In this early stage too, the pneumatocele may not be too obvious on the chest radiograph; unless areas of relative hypertranslucency and poorly defined crescentic lines representing the pneumatocele wall are constantly looked for, they are easily missed. It is questionable whether subpleural blebs are ever discernible on the chest radiograph, but they are readily seen by thoracoscopy once a pneumothorax occurs.

Seeing that in pneumatocele disease the parts of the lung not involved by the process may appear quite normal, and bear no resemblance to the blown-up spongy lung seen in diffuse emphysema, it may be well to refer to the bullæ as "lung" or "pulmonary" bullæ rather than "emphysematous" bullæ. In other cases there may be an associated lobar or sublobar involvement by diffuse emphysema, and it is conceivable that both conditions may have a similar etiology, the end result varying with the stage or duration of the disease. However, it appears likely that in the early stages of pneumatocele disease, and especially in young people, effective surgical treatment is available, the progress of the disease may be arrested, and the prognosis is good. In diffuse emphysema, on the other hand, we can offer nothing better than palliation, and the prognosis is not good.

TREATMENT OF PNEUMATOCELES

The physician plays a pre-eminent role in the prophylaxis and diagnosis of this disease, as well as in the palliative treatment of advanced cases.

On the basis of our present knowledge, the best prophylaxis is control of cough by removing its causes, as, for example, excessive smoking and chronic infection of the bronchi or upper respiratory passages, by the judicious use of cough sedatives, and by encouraging the patient to control cough voluntarily, through an intelligent explanation of the uses and abuses of this function. Seeing that cough is known to be a prominent and sometimes the only symptom of obscure but serious lung disease, such as carcinoma, tuberculosis, and the condition under discussion, it is evident that no doctor should encourage patients' acceptance of "only a cigarette cough". Careful physical examination, but above all chest radiography and, in case of doubt, consultation with a specialist in chest disease, is the only policy that will avoid disaster.

The surgeon is often called to see a patient with pneumatoceles only after a spontaneous pneumothorax has occurred, or after the pneumothorax has failed to respond to repeated air aspiration with needle and syringe.

It is evident from the foregoing that every non-tuberculous spontaneous pneumothorax, including those that tend to respond to air removal by syringe or even to simple bed rest, should be viewed as a probable indication of pneumatocele disease. At times, one or more pneumatoceles may be evident on the radiograph showing the pneumothorax, but whether this is so or not, it is thought wise to subject every spontaneous pneumothorax to thoracoscopy. This simple examination, done under local anæsthesia, will usually provide useful information as to the condition present and its management.

Sometimes at thoracoscopy no evidence of bullæ or blebs can be found. This is probably because the inflated pneumatocele which ruptured to cause the pneumothorax is now deflated and collapsed and hence no longer identifiable, or the pneumatoceles are on the mediastinal or diaphragmatic surface of the lung beyond the visual reach of the thoracoscope. Much more commonly, however, one or more pneumatoceles will be found if the lung surface is painstakingly searched, using two or more intercostal openings for the thoracoscope. Frequently a bulla will appear partly as completely deflated and attached at one point to the chest wall. Such a structure is apt to look like an adhesion on the radiograph and is often so interpreted. The type of pneumatocele found will determine the line of treatment to be followed.

As far as the subpleural blebs are concerned, it is the practice of the writer to touch them in several places with the electrocautery in an endeavour to destroy their wall. This done, two medium-sized catheters are placed through the intercostal openings for the thoracoscope, one catheter extending up into the dome of the

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pleura and the other resting on the diaphragm. Continuous suction at 12 to 20 cm. of water, depending on the rate of air leak, is then applied to the catheters. Each day a solution of penicillin, 100,000 units in 10 c.c. of normal saline, is injected through each catheter. Close supervision of lung expansion is exercised by daily radiography for the first three days, and then by radiography every two or three days. After seven to ten days, provided the lung is completely or almost completely expanded, the catheters are removed. In the case of patients with low respiratory reserve, particularly elderly patients, an emergency intercostal catheter set is kept ready in the room for a week or so after the catheters have been removed, because reopening of an air leak, perhaps by coughing, may lead to a dangerous tension pneumothorax with little or no warning.

It is necessary to use at least two catheters because, if only one is employed, the lung may expand around the catheter opening, sealing it. Should the air leak be occurring from another part of the lung, the pneumothorax will then persist.

The penicillin solution is injected for two reasons. It diminishes the chance of pleural infection, and the reaction caused by its pooling on the diaphragm encourages a creeping out and adherence of the lung, commencing at the base. Strong irritants such as silver nitrate, or coagulants, have been advocated to cause adhesions, but they have not seemed to the writer necessary or indeed desirable.

If definite bullæ encroaching on the lung body are found at thoracoscopy, it is probably best to resort to thoracotomy. With the chest open, the whole lung can be carefully palpated and all areas of bullous formation can be excised.

Often the bullæ are limited in extent, and lie at the periphery of a lobe. These can be isolated with clamps and removed, the clamped edge being oversewn in order to prevent air leak or hæmorrhage. If the bullæ are larger, extending deeply into the lobe, it is the practice of the writer to strip the pleural wall of the bulla away, and to ligate the points of air leak or bleeding. The denuded lung is left uncovered and becomes adherent to, and sealed by, the parietal pleura, when the lung is maintained expanded by catheter suction.

Formal lobectomy is necessary only in the occasional case, where a lobe has been destroyed by bullæ, or a combination of bullæ and diffuse emphysema involves the whole lobe. It would be well to refer those patients for surgical treatment as soon as bullæ are detected, and before complications such as pneumothorax or more serious lung destruction with corresponding respiratory insufficiency have developed.

Many cases of advanced disease, with large bilateral bullæ complicated by chronic bronchitis and chronic anoxia, are no longer within the scope of surgical aid, and must be treated by cough sedation, expectorants, bronchial relaxants and oxygen as indicated. Bouts of acute bronchitis or pneumonitis will call for antibiotics, after sputum culture has identified the predominating organisms.

It is a striking peculiarity of this disease that many patients who seem hopeless surgical risks at the commencement of thoracotomy become restored to nearly normal physiological balance as soon as the chest is opened and the embarrassing bullæ are punctured and deflated.[2] Anæsthesia up to this point may be tricky because exaggerated breathing due to anxiety or particularly forcible bagging by the anæsthetist may cause very marked inflation of the bullæ, with heightened pressure effects. For the same reason, patients with bilateral bullæ may present difficult anæsthetic problems, because while the surgeon can deflate the bullæ of the side opened, those of the other side cannot be so relieved.

SUMMARY

A short description of pulmonary pneumatoceles is given with a plea for their prophylaxis, early detection and treatment, in order to avoid the lung destruction which they may cause.

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PAPILLARY TUMOURS OF THE CHOROID PLEXUS*

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ALTHOUGH comparatively rare, papillary tumours of the choroid plexus are well known, and as a group they have been recognized as formidable neoplasms. A comprehensive review of the literature was made by Van Wagenen[1] in 1930. Including his two cases, 47 were recorded up to that time. Posey[2] reviewed the literature in 1942 and presented one case, bringing the total up to 86. Well over 100 cases had been recorded by 1954.

It is our purpose to record two cases: one in an infant in whom the tumour metastasized by way of the cerebrospinal fluid, and one in an adult which was surgically removed.

CASE REPORTS

CASE 1. M.M., an 11-month-old white male, was admitted to the Children's Hospital in Halifax on May 26, 1953, because of increasing irritability, restlessness and vomiting. The family history and past personal history were not unusual. The infant appeared normal at birth and had developed normally in every way until the onset of this illness. Early in May the child became irritable and restless, and began to vomit after meals. A physician was summoned and the infant was admitted to a hospital near the home of the parents. He appeared acutely ill at this time. His neck and back were rigid and Brudzinski's sign was positive. The cranial nerves, pupils, fundi, fontanelles and reflexes were normal.

Initially the spinal fluid contained increased protein but showed no other abnormality. Examination of the peripheral blood showed a white cell count of 14,950 with a normal differential. Five days later the C.S.F. pressure was 340 and analysis revealed protein 88 mg. %, sugar 64 mg. %, chlorides 650 mg. % and 4 cells per c.mm. Tuberculous meningitis was suspected, although the temperature had remained normal and the tuberculin patch test was negative.

On May 26, 1953, he was transferred to Halifax. At that time he was well nourished, apparently blind, very irritable, and obviously in pain when any attempt was made to flex the neck or back. The eyes were wide and staring. The pupils reacted sluggishly to light and the disc margins were indistinct. The anterior fontanelle was open and bulging. Brudzinski's and Kernig's signs were positive.

Investigation. Temperature 98.8° F. on admission; tuberculin patch test negative; Hb. 14.3 g.; white cell count 17,250 (neutrophils 28%, lymphocytes 61%, monocytes 3%, eosinophils 7%, basophils 1%); urinalyses negative; throat and nasal cultures negative. The C.S.F. pressure was increased and analysis revealed protein 100 mg. %, sugar 55 mg. %, chloride 750 mg. %, numerous red cells and 10 white cells (mononuclear) per c.mm.; cultures were sterile after 24 hours' incubation. A skull radiograph showed widely separated sutures. A ventriculogram disclosed a moderate degree of hydrocephalus involving the entire ventricular system. There was no displacement of the ventricles and no evidence of tumour. It was felt that a communicating hydrocephalus was present, possibly congenital in origin.

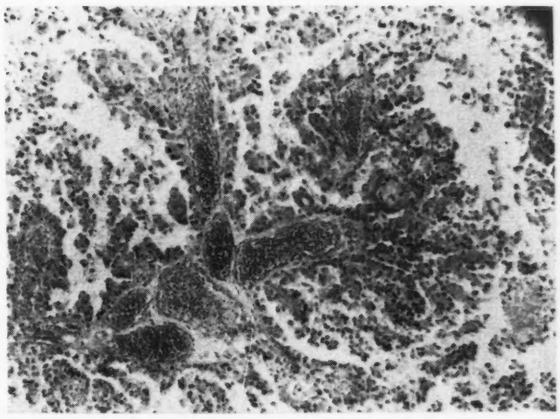

Fig. 1.—The cellular and highly vascular papilloma from Case 1. (× 110)

Progress. From the time of the ventriculogram until death he ran a high remittent temperature. Four days after the air studies a lumbo-peritoneal shunt operation was performed. For several days there appeared to be some improvement. Within a week, however, he began to have convulsions and soon these became almost continuous. Death occurred on June 9, 1953, approximately five weeks after the onset of his illness. (Cultures of the cerebrospinal fluid were negative for tubercle bacilli after eight weeks' incubation.)

Autopsy findings. The thoracic and abdominal organs showed no unusual features. *Cranial cavity:* The suture lines were grossly widened. The pia-arachnoid at the base of the brain was of a dull white colour and was flecked by very numerous small yellow thickenings. The meninges over the vertex and cerebellum appeared grossly normal. The appearances were suggestive of tuberculous meningitis. There was a marked degree of hydrocephalus involving the entire ventricular system. The choroid plexus of the left lateral ventricle was largely replaced by a hæmorrhagic papillomatous tumour measuring approximately 2 x 1 cm. There was no evidence of metastatic involvement of the lining of the ventricles or aqueduct.

Histology. Sections of the tumour showed a cellular and highly vascular papilloma (Fig. 1), which on the whole was well differentiated and uniform. The connective tissue cores of the papilloma were covered by one to several layers of cuboidal to low columnar cells and in many areas the cells appeared to be breaking

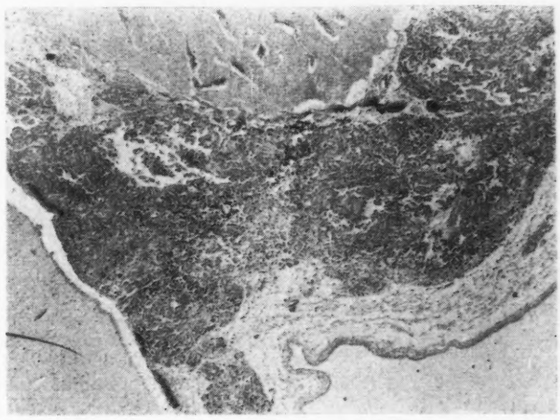

Fig. 2. (Case 1).—Involvement of the meninges by tumour. (× 50)

*From the Institute of Pathology of Dalhousie University and the Province of Nova Scotia, Halifax, N.S.

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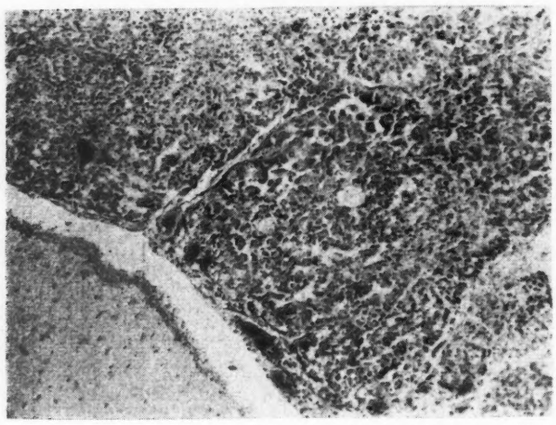

Fig. 3 (Case 1).—An area of cellular pleomorphism in the tumour in the meninges. (× 110)

away (Fig. 1). Cilia and glial elements were absent and there was no sign of calcification. Sections also revealed widespread involvement of the meninges over the base of the brain, vertex and cerebellum by the tumour (Fig. 2). This accounted for the yellow thickenings observed at autopsy. In the meningeal metastases there were occasional areas of pleomorphism (Fig. 3). There was no evidence of invasion of the grey matter, and the lining of the other ventricles and aqueduct was free from tumour.

CASE 2. U.M., a 31-year-old married white woman, was admitted to the Victoria General Hospital in Halifax on April 2, 1954, complaining of progressive loss of vision associated with occasional morning headaches during the previous five months. On a few occasions she had suffered from slight dizziness.

Examination revealed bilateral papillœdema, which was slightly more marked on the right side. Reflexes were normal and there were no nystagmus, sensory or motor loss, or cranial nerve signs. Blood pressure was 130/80 mm. Hg. Examination of the heart and chest was negative.

Investigation. E.E.G. normal; chest radiograph negative; C.S.F. chlorides 670 mg. %, protein, sugar and cell count normal. A ventriculogram revealed dilatation of both lateral ventricles and the third ventricle. There was no evidence of ventricular shift. Special studies of the fourth ventricle showed it to be displaced backwards and there was evidence of a bulging tumour mass on the anterior wall.

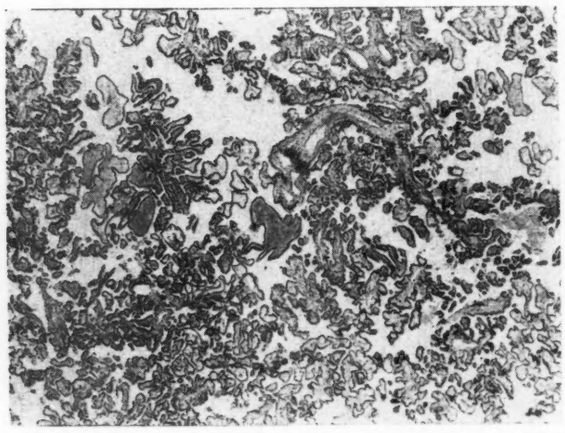

Fig. 4 (Case 2).—Showing the typical structure of a choroid plexus papilloma. (× 50)

At operation (Dr. W. D. Stevenson), the fourth ventricle was entered and a papillomatous tumour was removed.

Progress. Her postoperative course was uneventful and she was discharged in good condition on May 5, 1954. A follow-up in February 1955 revealed that the patient had had no further symptoms.

Pathology. The surgical specimen consisted of a rather friable papillomatous tumour measuring approximately 4 x 2.5 cm. Sections showed a typical choroid plexus papilloma (Figs. 4 and 5) consisting of vascular connective tissue cores covered by a single layer of columnar epithelium. The surface epithelium showed no evidence of heaping up and was everywhere uniform. Cilia, glial elements and calcium were again absent.

DISCUSSION

Papillary tumours of the choroid plexus have been reported in all age groups up to the eighth decade, but the highest incidence is encountered in the first decade.[2] Drucker[3] has reported a case of choroid plexus papilloma in a stillborn infant. In infancy the tumours are encountered most commonly in the lateral ventricles and there has

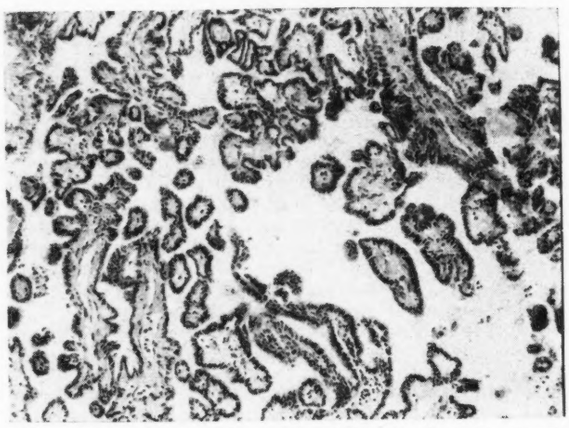

Fig. 5 (Case 2).—Higher magnification showing simple structure and good differentiation. (× 110)

been a curious preponderance on the left side.[1-3] In adults the commonest site is the fourth ventricle.[2] Hydrocephalus is commonly met with in the young, but is less frequently encountered in adulthood.[4]

The clinical picture may be quite obscure or suggestive of some other disease. If a tumour arises in a lateral ventricle, for example, it may reach a considerable size before causing any trouble, and only a complication such as meningeal involvement or hydrocephalus may produce symptoms. In our Case 1 the diagnosis for some time was considered likely to be tuberculous meningitis, and a similar diagnosis was considered by Van Wagenen[1] in his Case 2. Analyses of the C.S.F. in reported cases have yielded variable results, and this was so in the cases

described here. Attention has been drawn by several writers[11, 12] to the identification of tumour cells in the C.S.F. However, no one has yet been able to identify cells from a papillary tumour of the choroid plexus during life. In the cases reported here the identification of tumour cells in the C.S.F. was not attempted.

In view of the frequency of associated hydrocephalus,[2] one should keep these tumours in mind in the differential diagnosis of hydrocephalus in infancy. This was mentioned by Posey[2] and has recently been re-emphasized by Lassman.[4] The pathology of hydrocephalus with choroid plexus tumours is controversial. Kahn and Luros[5] feel that their Case 1 proves the production of hydrocephalus by oversecretion of C.S.F. by the papillary tumour, but in most recorded cases mechanical block seems to be the most probable explanation. In our Case 1 the possibility of mechanical blockage cannot be excluded. In our Case 2 the hydrocephalus was certainly attributable to obstruction by the tumour.

The signs of meningitis in our first case were no doubt due to the diffuse involvement of the meninges by tumour. Metastatic involvement of the meninges ("seeding") has been previously recorded in several cases.[6-9] Although the propriety of referring to such meningeal involvement as metastasis has been questioned,[10] it has been clearly pointed out by Willis[9] that any distinction between "seeding" and metastasis is needless. A tumour need not be histologically malignant in order to metastasize.

With regard to therapy, there have been good results from surgical intervention in a few cases[2, 5] but the outlook is not especially favourable. The principal reasons for this would appear to be the difficulties in early diagnosis, complications such as meningeal metastases, technical difficulties created by the bulk or location of the tumour and recurrences. Posey[2] reviewed 22 cases in which the tumour was surgically attacked, and in only five was there complete surgical recovery. More recently Kahn and Luros[5] reported on seven patients treated surgically among whom five had died, one was alive and well five years postoperatively, and one was alive one year postoperatively.

In our second case the patient is free of symptoms nine months postoperatively, but it is too early yet to estimate the likelihood of cure.

SUMMARY

Two cases of papillary tumour of the choroid plexus have been recorded and some of the general characters of such tumours have been discussed.

In one case in an infant the tumour, although histologically simple, gave rise to widespread meningeal metastases via the C.S.F. with the production of a "tumour meningitis". In the second case the tumour was surgically removed and the patient is alive and well nine months later.

Attention has been directed to the necessity of considering these tumours in the differential diagnosis of hydrocephalus, particularly in the young, and also in cases of clinically atypical meningitis.

The authors gratefully acknowledge the kind assistance of Prof. N. G. B. McLetchie.

Thanks are also due to Dr. G. B. Wiswell and Dr. W. D. Stevenson for permission to publish their cases.

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PROSTATIC CARCINOMA: TREATMENT WITH DIETHYLSTILBŒSTROL DIPHOSPHATE

Diethylstilbœstrol diphosphate is a well-tolerated œstrogen for intravenous use which R. H. Flocks and co-workers used in a series of 66 patients with prostatic carcinoma; 34 had failed to respond or had become refractory to the usual œstrogens. The dose ranged from 250-1,250 mg., in an intravenous infusion of saline or 5% glucose. The rate of administration averaged 300 c.c. per hour, and usually one or two infusions were given daily. A course ranged from five to 20 days, depending on clinical response. Courses were repeated monthly. Thirty-eight patients had only a single course; the highest total dose was 31,500 mg. Side-effects were slight, mainly nausea, dizziness and perineal burning.

Only one case showed demonstrable evidence of improvement of metastases; 12 showed progression of bony metastases. The prostate softened or shrank in seven cases. Bone pain improved in 27 out of 32 cases. Urinary symptoms improved in 15 out of 27 and there was increased well-being in 32 out of 46 cases.—R. H. Flocks *et al.*: *J. Urol.*, 74: 549, 1955.

SEIZURES OCCURRING DURING INTENSIVE CHLORPROMAZINE THERAPY*

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WE HAVE NOTED the occurrence of 14 convulsive seizures in 11 patients out of a group of 21 given intensive chlorpromazine therapy (up to 4,000 mg. per day). We believe this high incidence justifies a preliminary report.

Prior to the start of our investigation into the effectiveness of intensive chlorpromazine therapy for psychotic patients we had treated 364 patients with chlorpromazine between April 12, 1954, and August 31, 1955. The doses used ranged from 25 mg. to 600 mg. per day. More than 95% of the drug administration was by the oral method. The individual dose was never higher than 300 mg.

We encountered, among other side-effects, seizures in one patient. This 48-year-old woman had five generalized convulsive seizures while receiving 100 to 200 mg. chlorpromazine given daily in four divided doses. She had a history of convulsive seizures of the same type in 1952, two years after bilateral prefrontal leukotomy.

Several authors have reported the occasional occurrence of convulsive seizures. Lehmann[1] describes seizures in patients receiving chlorpromazine and with a history of convulsive disorders. He also reports three cases without previous epileptic manifestations in which tonic and clonic phenomena almost amounting to a grand mal seizure were observed. He concluded that cerebral hypoxia was responsible for this phenomenon, as the convulsions occurred during syncopal attacks due to orthostatic hypotension.

Kinross-Wright[2] reported the occurrence of grand mal seizures in a case of chlorpromazine therapy, with no previous history of convulsive disorders, while in some cases he found evidence that chlorpromazine had anticonvulsant activity. Goldmann,[3] in a series of 500 patients receiving chlorpromazine in doses up to 2,400 mg. per day, reported no seizures. Winkelman[4] has found evidence that chlorpromazine increases the effectiveness of anticonvulsants.

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THE INVESTIGATION

Our main investigation was designed to measure the psychiatric effect of intensive chlorpromazine therapy on selected psychotic patients. A paper discussing methods of treatment, side-effects, complications and results is in preparation.

This present report on seizures is intended to make as many facts as possible available to other investigators for use in their studies. Consequently we do not attempt any detailed interpretation or discussion of our findings.

E.E.G. RECORDS

Reports of the sporadic occurrence of convulsive seizures during chlorpromazine therapy led us to include routine pre-treatment, treatment and post-treatment electroencephalograms (E.E.G.) as part of our procedure. The actual

TABLE I.

Case	*Sex and age*	*Admissions to mental institution*	*Diagnosis*	*Previous psychiatric treatment*
Case 1	F. 26	1948 1950	Schizophrenic, catatonic type	E.S.T.: 1950 I.S.T.: 1950 E.S.T.: 1951, 52, 53. Leukotomy 1955
Case 2	F. 31	1951	Schizophrenic, catatonic type	E.S.T.: 1951 I.S.T.: 1951 E.S.T.: 1952-55
Case 3	F. 40	1945 1953	Schizophrenic, simple type	E.S.T.: 1945 I.S.T.: 1948 E.S.T.: 1949 E.S.T.: 1955
Case 4	F. 26	1955	Schizophrenic, paranoid type	I.S.T.: 1955 Chlorpromazine Small doses 1955.
Case 5	M. 25	1949	Schizophrenic, catatonic type	E.S.T.: 1949 Serp. 54. I.S.T.: 1949 Leukotomy 1952 E.S.T.: 1952
Case 6	F. 32	1954	Schizophrenic, paranoid type	Chlorpromazine 150 mg. daily 1955. E.C.T.: 1955 M.E.R. 17, 1955 Craniotomy July 1954
Case 7	F. 44	1941	Schizophrenic, paranoid type	I.S.T.: 1941
Case 8	F. 25	1943	Schizophrenic, hebephrenic type	E.S.T.: 1943 I.S.T.: 1944 E.S.T.: 1949 Leukotomy 1950, 1955
Case 9	M. 46	1943	Schizophrenic, paranoid type	I.S.T.: 1943 E.S.T.: 1943, 48 Leukotomy 1952 Chlorpromazine 1954, 100 mg.
Case 10	M. 31	1942	Schizophrenic, simple type	I.S.T.: 1942 Leukotomy 1950 E.S.T.: 1951-54 Chlorpromazine 1954 E.S.T.: 1955
Case 11	F. 50	1935 1942	Schizophrenic, other and unspecific.	E.S.T.: 1942 I.S.T.: 1942 Chlorpromazine 1955, 150 mg.

E.S.T. = Electroshock therapy.
I.S.T. = Insulin shock therapy.

TABLE II.

Case	*Convulsive disorders in fam. hist.*	*History of spontaneous seizures*	*Previous E.E.G. indicative of ictal disorder*	*Seizures during insulin shock therapy*	*Seizures after leukotomy*	*Remarks*
Case 1	Negative	Negative	1955: E.E.G. suggestive of ictal disorder	No	No	
Case 2	Negative	Negative	No	No	—	
Case 3	Negative	Grand mal seizures 1950-1951.	1950: E.E.G. typical of idiopathic epilepsy.	No	—	
Case 4	Negative	Negative	—	Grand mal seizure on 280 u. insulin.	—	
Case 5	Negative	Negative		3 grand mal seizures on 330 u. insulin.	1 g.m. seizure.	
Case 6	Negative	Negative	Jan. 1955. Rhythmic slow patterns in temporal area.	—	—	Craniotomy 1954 Postoperative convulsion.
Case 7	Negative	Negative	—	No	—	
Case 8	Negative	Negative	No	1944: 1 grand mal seizure on 300 u. insulin.	No	
Case 9	Negative	Negative	No	No	No	1944 elevation of depressed fracture of rt. frontal area.
Case 10	Negative	Negative	No	No	No	
Case 11	Negative	Negative	—	No	—	

occurrence of seizures necessitated a post-seizure E.E.G. as soon as possible. Dr. M. Saunders, Winnipeg, our consulting electroencephalographer, has agreed to report his findings in a separate paper.

PATIENTS WHO HAD SEIZURES

Eight of the patients who had seizures were women aged 25 to 50 and three were men aged 25 to 46. All were diagnosed as schizophrenic.

Admissions to hospitals, diagnostic subtypes and previous treatments are given in Table I. None of the cases had a definite family history of convulsive disorders (Table II.) One had a history of spontaneous seizures, with the E.E.G. typical of idiopathic epilepsy. One had E.E.G. findings suggestive of ictal disorders but no history of convulsions. Ten of the 11 cases had had previous insulin shock therapy; two of them had one seizure each during the treatment; one patient had three seizures. Four patients had prefrontal leukotomies, post-leukotomy seizures occurring in one patient. Case 6 had a previous postoperative convulsion after craniotomy in 1954. Case 9 underwent a cranial operation for elevation of a depressed fracture in the right frontal area in 1944, with no preoperative or postoperative seizures reported.

All patients who had seizures though taken off treatment for 24 hours were subsequently treated with high doses of chlorpromazine—in some cases higher than those at which seizures occurred.

RELATION OF SEIZURES TO DOSES (TABLE III)

In ten cases, the seizures occurred within an average of two hours after an oral dose of 400 to 600 mg. chlorpromazine. In Case 5 a convulsion occurred four hours after a dose of 100 mg., and a second time 10 minutes after a dose of 100 mg.

The daily dose before the occurrence of seizures ranged from 200 to 2,900 mg., the number of treatment days up to the time of convulsion varied from 3 to 36 days, and the total dosage up to the time of the seizures varied

from 500 mg. to 60,900 mg. chlorpromazine (Table III).

TABLE III.

RELATION OF SEIZURES TO DOSAGE

	Last dose before seizure			*Day of seizure*	*Total dose before seizure*	
	Dose	*Time given*	*Time of seizure*	*Total dose before seizure*	*Days of treatment*	*Total dose given*
Case						
1	600 mg.	4 p.m.	5.50 p.m.	1,800 mg.	10 days	10,500 mg.
2	600 mg.	4 p.m.	6.10 p.m.	1,800 mg.	10 days	10,500 mg.
3	400 mg.	4 p.m.	5.55 p.m.	1,200 mg.	18 days	20,100 mg.
4	600 mg.	12 noon	1.20 p.m.	1,200 mg.	7 days	7,500 mg.
5 A	100 mg.	8 a.m.	2.15 p.m.	200 mg.	3 days	500 mg.
B	100 mg.	9 p.m.	9.10 p.m.	700 mg.	7 days	10,700 mg.
6	500 mg.	8 p.m.	8.30 p.m.	1,800 mg.	18 days	43,500 mg.
7	600 mg.	4 p.m.	5.30 p.m.	1,800 mg.	10 days	10,500 mg.
8	600 mg.	12 noon	2.45 p.m.	1,200 mg.	10 days	10,500 mg.
9	600 mg.	12 noon	1.10 p.m.	1,300 mg.	9 days	11,200 mg.
10	600 mg.	6 p.m.	7.45 p.m.	2,900 mg.	24 days	45,500 mg.
11 A	600 mg.	8 a.m.	11.30 a.m. 1.30 p.m.	600 mg.	8 days	11,800 mg.
B	600 mg.	12 noon	2.00 p.m.	1,000 mg.	19 days	23,400 mg.
C	400 mg.	2 p.m.	2.10 p.m.	1,500 mg.	36 days	60,900 mg.

DESCRIPTION OF SEIZURES

The following is a description of five of the 14 seizures as observed by the nursing staff, the seizures ranging from loss of consciousness with few convulsive phenomena, to generalized tonic, clonic convulsions with evidence of autonomic activity.

CASE 7

Patient was seen falling, tried to save herself from fall. Her body was quite lax. Patient appeared pale, clammy, with no twitching, only her head moving slightly. Some drooling present, no tongue biting, no incontinence. Felt quite drowsy afterwards. Patient remembers falling, but had amnesia for several minutes.

CASE 2

Fell, stiffening, rigid, striking head. Appeared cold and clammy. Hands moderately twitching. Little salivation. Very mild cyanosis. No incontinence. After 10 minutes would answer questions but did not remember. Amnesia for a period, from just before seizure to about 30 minutes after seizure. After this, patient stated she felt wide awake, and appeared more alert than during the day.

CASE 3

Patient became limp suddenly and was very pale, cold, sweating; pulse rapid and faintly perceptible. Generalized very mild convulsions. Had amnesia for some time before seizure until shortly after seizure. Still felt tired one hour afterwards. No period of excitement.

CASE 9

Patient was seen walking, told attendant that his legs felt weak, then collapsed and became unconscious. Attendants who carried him to bed noticed that his legs were limp. On the bed he went into clonic convulsions, which were generalized and lasted for about 30 seconds, without preceding tonic phase. During seizure patient was cyanosed; pulse fast. No incontinence, no frothy saliva. Cyanosis cleared quickly. Unconscious for 10 minutes. No excitement stage after the seizure.

CASE 11

Patient fell with a cry and immediately developed tonic, clonic convulsions, lasting about one minute. Her colour was first cyanotic, then ashen. Pulse was faint and irregular. Patient was incontinent during seizure. Semi-comatose state lasted for about one hour, during which patient was very quiet. Had amnesia during the convulsion.

The remaining nine seizures were similar to those described. Before the third seizure in Case 11, myoclonic fits were observed.

SUMMARY AND CONCLUSIONS

The occurrence of 14 seizures in 11 patients out of a group of 21 psychiatric cases treated with high doses of chlorpromazine is reported. Some of the cases had a history of convulsive disorders, the seizures occurring spontaneously, during insulin shock therapy or after leukotomy. In some patients the convulsive seizures occurred for the first time while on intensive chlorpromazine therapy. In nine of the 11 cases the seizures occurred in the second treatment week after a total dosage of 10,000 mg. chlorpromazine. There seemed to be a time relationship between seizure and the last preceding medication.

From a clinical standpoint the seizures are thought likely to be of the type occurring with syncopal attacks due to cerebral hypoxia, which is in agreement with the conclusion of Lehmann.[5] An exception is the third seizure in Case 11 (Table III) which seemed to be due to toxicity.

These conclusions based on clinical observation alone may have to be revised following further study of the E.E.G changes found in practically all cases during intensive chlorpromazine therapy.

We are indebted to Poulenc Limited, Montreal, for generous supplies of chlorpromazine (Largactil) for this investigation.

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ACUTE APPENDICITIS COMPLICATING PREGNANCY

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ACUTE APPENDICITIS is not a frequent complication of pregnancy but it is a serious one, particularly in the last trimester and whenever diagnosis and treatment are delayed. Very little has been published on this subject in the past 10 years, since the introduction of our modern advances in surgery. The literature prior to this era presents impressively high figures for maternal and fetal loss, and also some alarming recommendations on treatment. A study of the available recent literature, plus the results in the present series of 20 cases,* indicates that both maternal and fetal loss need rarely occur, provided that the appendicitis is treated promptly and the pregnancy left alone.

In this series, five of the operations were elective appendectomies for recurrent appendiceal colic in pregnancy. All five cases progressed uneventfully to normal delivery. In another 14 cases, the abdomen was opened as an emergency for acute appendicitis; acute suppurative disease of the appendix was encountered and the appendix was removed in seven of these cases. In two of the other seven cases markedly acute mesenteric lymphadenitis was encountered. In the remaining five, pathological examination did not show acute inflammation. Appendectomy in each of these cases relieved the patient's complaints.

In the remaining case, the first of the series, an acutely inflamed appendix was left by me because of misdiagnosis, with resultant abscess formation and prolonged morbidity. Apart from this case of abscess formation, the only other complication occurred in the second case—an acutely inflamed appendix in a woman four months pregnant which I left overnight because of uncertainty, and in a misguided attempt to "give the patient a chance". The fetus in this case was passed dead two weeks after operation.

INCIDENCE

The incidence of acute appendicitis complicating pregnancy is 0.07%, or seven cases in 10,000 deliveries. This figure is obtained from reported cases occurring in half a million deliveries.

RELATIONSHIP TO GESTATION

It would appear that 70 to 80% of cases of acute appendicitis complicating pregnancy occur within the first six months. Certainly it is true that the condition is rare as a complication of labour or the puerperium. Norton and Connell in the Mayo Clinic collected 1,110 cases from the literature, with only nine cases occurring in labour or the puerperium. De Lee mentions five cases. Meiling in 1947, in a large series, reported two in labour and one in the puerperium. In my series, of the eight cases with acute suppurative disease, three occurred at about eight months, two occurred at six months and the remaining three occurred at between three and four months.

The only possible explanation which I can give for this interesting point is a mechanical one related to the change in position of the appendix. Baer and co-workers made radiographic studies in 78 cases throughout pregnancy. Progressive displacement of the cæcum and appendix upward was demonstrated in a certain percentage of cases. The appendix in the average case was visualized at the level of the iliac crest at the fifth month. At the seventh month, in 88% the appendix was observed to have risen above the level of the iliac crest. There was return to normal position by the tenth post-partum day.

To correlate these observations, let us say that, broadly speaking, we recognize two main types of appendicitis—obstructive and infective. The obstructive type develops on the basis of adhesions, peritoneal bands, or fæcoliths. The day arrives when, because of the obstructing mechanism, the blood supply to the organ distal to the obstruction becomes insufficient, with obvious sequelæ. If a woman has an appendix in which the field is fertile for obstructive appendicitis, this would most probably occur at the stage of pregnancy when the appendix is being pushed around from the third to the sixth month.

It is interesting that in three of my cases inflammation occurred at eight months—once with perforation. All three did well, although one patient delivered one week after the appendectomy, in a remarkably normal manner and without complication.

Under the same heading I would like to mention another interesting case of acute abdominal

*These patients came under my care as surgeon on the staffs of the Montreal General and Reddy Memorial Hospitals, and as consultant in surgery at the Catherine Booth Mothers Hospital, Montreal.

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disease in pregnancy, not included in this series. This patient was four months pregnant and had acute small bowel obstruction. She had had an appendectomy about three years before. One year previously, also at four months in pregnancy, she had had a similar attack of intestinal obstruction, which was relieved by intubation decompression. Operation revealed a complete obstruction of a loop of small bowel by an adhesive band in the appendiceal area. Thus on two occasions, both at four months in pregnancy, this patient developed small bowel obstruction, presumably due to the mechanical displacement of bowel by the enlarging uterus.

DIAGNOSIS

The first point to be discussed is awareness. Awareness of the possibility is the sheet anchor in the diagnosis of this condition, as it is in so many diseases.

As pregnancy advances, abdominal distension and discomfort, flatulence, constipation and similar symptoms may develop, and unless the obstetrician remembers the possibility of coexistent appendiceal disease, it is easy to overlook its presence until great harm has been done. It is also well to remember that the percentage of atypical cases of acute appendicitis is high at all times and higher than usual in pregnancy, because the disease is modified by the coincident presence of the gestation.

Coincidental disease is typically one of the physician's most common and dangerous pitfalls. I have seen the statement borne out on two occasions that one of the most dangerous places to develop appendicitis is in a hospital. When a patient in the ward with a fractured leg or pneumonia complains of nausea and vomiting, it is so easy to attribute it to the medication or something else, until finally the evidence of the patient's temperature chart, vomit pail, and complaints can no longer be ignored.

Although a feature of appendicitis is the atypical case, the vast majority of histories follow a very typical pattern, which might be called a triad of events. (1) First there is the sudden onset, frequently preceded by a period of varied gastrointestinal symptoms. (2) Secondly, there are crampy pains projected over an area of the abdomen and usually associated with some degree of nausea and/or vomiting; this can be described as the peristaltic phase of the disease. These cramps usually cease before the third stage is fully developed. (3) The third phase is the localization of a steady dull pain definable by the tip of the patient's finger, and may be termed the phase of peritoneal irritation.

With few exceptions, this triad indicates the presence of appendicitis, regardless of any contrary physical or laboratory findings. On physical examination the sign of tenderness, distinctly separated from the symptom of pain, occurring on the right side of the abdomen, is the cardinal feature in the diagnosis of acute appendicitis at any time. The white cell count may or may not give information, but should certainly be done in all cases of suspected appendicitis. The consideration of Virchow's "physiological leukocytosis of pregnancy" can only lessen the importance of the findings in this laboratory procedure.

DIFFERENTIAL DIAGNOSIS

If at the conclusion of this presentation I have succeeded in showing that appendicitis is the first possibility in the study of right-sided distress in pregnancy, I shall have achieved my purpose. Differential diagnosis is from several obstetrical and gynæcological conditions which I wish only to mention, such as torsion of an ovarian cyst, fibroid degeneration, and ectopic pregnancy. Acute or subacute pyelonephritis is so frequently difficult to diagnose, the main features being, of course, chills and pyuria. Mesenteric lymphadenitis cannot be distinguished from acute appendicitis until the abdomen is opened. One can have hunches, but in my opinion one would be foolish to spare the knife with this diagnosis in mind. In one of my cases this disease was unusually severe in nature, the glands being distended with a hæmorrhagic extravasation.

TREATMENT

Once the diagnosis of acute appendicitis complicating pregnancy is made, the treatment recommended is appendectomy. Procrastination will lead only to an increased rate of fetal loss and increase in maternal morbidity. The pregnancy should be left alone.

It is recommended that the operation be performed under spinal anæsthesia, for two reasons: (1) to prevent interference with oxygenation of the fetus; (2) to lessen the occurrence of the violent excursions of abdominal contents caused by nausea and vomiting.

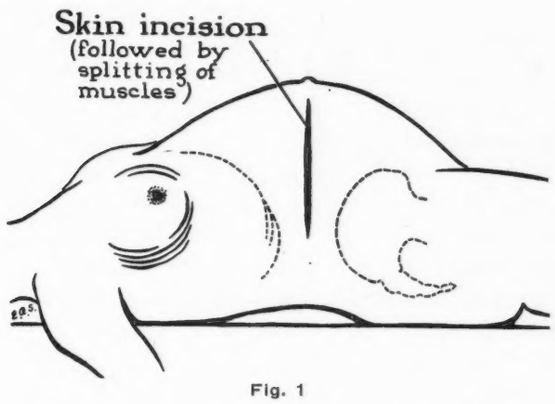

Fig. 1

It is also recommended that the incision be a long transverse skin incision at the level of the umbilicus, followed by muscle splitting as necessary. Most careful avoidance of the uterus and ligaments in the handling of retractors should be an obvious precaution.

Hormones as an adjunct to therapy were not used in any of these 20 cases, and their use is considered unnecessary. No other special feature, either in hospitalization or in convalescence, distinguished the care of these patients from that in appendicitis in the non-pregnant state.

In study of the literature it is evident that controversy has existed concerning the handling of the pregnancy in the presence of acute appendicitis. I do not wish to entertain any discussion on this, for me, dangerous and foreign field, because I am a general surgeon. I would close this subject with two remarkable quotations:

"The performance of radical obstetrical procedures in the presence of acute appendicitis does not seem to be good obstetrics or good surgery, and I would condemn it."[3]

"When one considers the high mortality of suppurative peritonitis at or near term, perhaps it would be best in the interest of the two individuals to remove the appendix and to do a Cæsarian hysterectomy at the same time."[1]

THE PROBLEM OF MATERNAL MORTALITY

As stated before, most of the literature on this subject antedates the general availability of antibiotics. In addition, one must consider other advances in surgery, such as hydration, intestinal decompression, and use of anticoagulants. A few of the more recent figures follow:

Irving: Boston Lying-In Hospital (1936): maternal mortality 11.8%.

Krieg: Four Detroit hospitals (1941): 200 cases—4 deaths—2% in the acute cases—maternal mortality 7.7%.

Cosgrove: Jersey City (1937): 18 cases—1 death—mortality 6%.

De Lee: "mortality in appendicitis complicating late pregnancy approaches 40%".

Marbury: (1933) 34 cases in third trimester—maternal mortality 26.4%.

Smith and Marbury: 3 deaths in 20 cases—mortality 15%.

These figures are startling, even if one considers only the lower ones of 6 and 7%, and when one considers mortalities of 15 and 26% one must realize that this is a very serious problem.

The reasons why perforation and suppurative peritonitis are much more serious in the pregnant state may be listed as follows:

1. Protective adhesions are less likely to be formed, the omentum and bowel being pushed away by the enlarging uterus.

2. Suppuration takes place higher in the abdomen. It is well known that the abdomen can handle suppuration much better in the lower than in the upper parts.

3. Bacteræmia can lead rapidly to infection of placental site, with not only abortion and its attendant dangers, but also another huge area of infection for the mother to combat.

4. Intestinal distension compromises the respiration sooner, because of the limited respiratory excursion in late pregnancy. This fact may lead to more frequent pulmonary complications.

THE PROBLEM OF FETAL LOSS

There are five main factors bearing on fetal loss: (1) the anæsthesia, (2) the operation, (3) the stage of the disease, (4) the disease itself, and (5) the stage of pregnancy.

It is my opinion, gathered from a study of the literature and current discussion, that anæsthesia plays but a very small part, if any, in the initiation of abortion or premature labour, provided that oxygenation of the fetus is not impaired and the course a smooth one.

It is difficult to understand how a properly executed operation could play any sizable part in this problem. I feel that if operation is reasonably well done one can disregard the mechanical factor. One must consider, only to mention, two other points: (a) absorption of products of wound healing, and (b) hormonal changes resulting from the patient's apprehension.

All figures show that the more advanced the disease, the higher the rate of fetal loss.

The presence of the disease itself plays the major part in the problem of fetal loss. Hoffman

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in 1949 presented figures relevant to this statement. He reported 47 cases of appendicitis in pregnancy, occurring in Detroit: 23 of these cases were of proven acute suppurative disease, and the other 24 were labelled "chronic" but had been diagnosed preoperatively as acute. In the cases with suppurative disease the fetal loss was 39.1%. This figure is subdivided into 23% early acute and 55% late acute. In all 24 cases which were not pathologically acute there was no fetal loss.

In the average pregnant woman with right lower quadrant pain suspected of being acute appendicitis, of short duration, what is the risk in recommending appendectomy, as against leaving the case under further observation? To answer this question, a study was made of the problem of abortion as a complication of operation in the pregnant woman. An average figure gathered from reports available on this subject is between 5 and 6% fetal loss.

From available literature, therefore, the risk of fetal loss when operation is performed on a case misdiagnosed as acute appendicitis should be less than 6%. The risk of fetal loss in early appendicitis with appendectomy approaches 15-20%, and this advances with the progress of the disease up to and above 50%.

CONCLUSIONS

1. The dangers and complications of acute appendicitis in the pregnant woman are those of the same condition in the non-pregnant woman, plus a confused clinical picture, delayed diagnosis, liability to general peritonitis rather than localization, and fetal loss.

2. Abortion and premature labour are not likely to occur if the disease is diagnosed promptly and appendectomy performed without delay.

3. From available reports on acute appendicitis, the risk of fetal loss starts at 15 to 20% and increases with the stage of the disease. With operation in cases of a mistaken diagnosis of acute appendicitis, the risk of fetal loss begins at 5-6%.

4. The fetal mortality is always high in advanced cases and is not improved by delay in surgery, which merely increases the maternal risk.

5. In the first trimester, recurrent right-sided abdominal pain at all suggestive of appendiceal colic warrants elective appendectomy.

6. The two major hazards to be overcome for improved results both centre about delay in diagnosis, which, as in all general reports on appendicitis, is the greatest single factor in morbidity and mortality. The first hazard is failure to recognize the possibility of this coincidental condition. The second hazard is procrastination. Leaving an acute appendix overnight will do nothing but increase the fetal loss.

7. The solution of the problem of acute appendicitis in pregnancy lies in the close co-operation of obstetrician and surgeon. The principles on which such a case is treated are threefold: that a woman with acute appendicitis has a dangerous disease, the risk of which is increased by the fact of her pregnancy; that the status of her pregnancy is not improved by any delay in handling the surgical condition; and that the treatment of acute appendicitis, in pregnancy as at any other time, is exclusively surgical.

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INTERNATIONAL SOCIETY OF HEMATOLOGY

Those interested are asked to note that the date of the 6th Congress of the International Society of Hematology has been moved forward one week to August 26—September 1 inclusive, while the convention site has been shifted to the Hotel Somerset, Boston, Mass. The 6th Congress of the International Transfusion Society will follow at the same hotel on September 3-5. The official languages will be English, French, Spanish and Interlingua. The scientific programme will include plenary sessions on leukæmia, nucleonics, spleen and reticulo-endothelial system, hæmorrhagic disorders, anæmia and immunohæmatology. Information may be obtained from the office of the President, Dr. William Dameshek, New England Center Hospital, Boston 11, Mass.

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Case Reports

MERCURY BICHLORIDE (CORROSIVE SUBLIMATE) POISONING

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MERCURY BICHLORIDE POISONING may occur from inhaling heavy concentrations of the substance in vapour or from its overuse in douches, but is more frequently due to ingestion, either accidentally or with suicidal intent. Although no Canadian figures are available, over 100 cases of mercury bichloride poisoning have been reported from Baltimore alone by Longcope and Luetscher.[1] This form of suicide attempt may not be as fashionable in Canada. To my knowledge, this is the first case of mercury bichloride poisoning reported in Canada, and is therefore of interest.

Patient J.H., aged 27 and an unemployed dairy worker, swallowed 1 g. of mercury bichloride about 11:45 p.m. on March 4, 1955, with suicidal intent. Shortly after, he developed severe abdominal pain and went to the nearby police station where, at about 12:40 and again at 12:50 a.m., he vomited blood and semi-formed whitish material. He was first seen in the Emergency Department of McKellar Hospital at 1:45 a.m. on March 5, 1955. He then complained of severe abdominal pain, was ambulatory and was retching continuously. He appeared robust, and weighed about 150 lb. His past history, aside from periods of mental depression, was negative. Temperature was 98.6° F., head and neck were normal, chest was clear, blood pressure was 118/70 mm. Hg, pulse 88 and of good quality. There were no cardiac abnormalities. He had generalized abdominal tenderness, most marked in the epigastrium, but when seen initially there was no rigidity or guarding of the abdomen and bowel sounds were normal. The vomitus contained shreds of tissue and altered blood.

The stomach was washed out with a concentrated solution of sodium bicarbonate until the return was clear, and Wangensteen suction instituted. He was also given 500 c.c. of plasma, 500 c.c. of whole blood, and intravenous fluids. Demerol was required and partially alleviated his severe abdominal pain. At 3:30 a.m. on March 5, after the nature of the poison ingested had been discovered, he was given 3.5 c.c. (350 mg.) of 10% BAL (dimercaprol) in 20% benzyl benzoate and peanut oil, deeply intramuscularly, and 1.75 c.c. (175 mg.) at 6:00 a.m. This latter dose was repeated every 4 hours for four doses, then every 8 hours for four doses, and finally every 12 hours for four additional doses to a total of 25.25 c.c. (2,525 mg.), all deeply intramuscularly in the gluteal region and without undue discomfort. At no time did he show any signs of side-effects or intolerance to this dose of BAL.

About 4:00 a.m. he began to pass very dark urine, with much sediment present and a trace of albumin. During the next few hours he also passed several slimy, bloody stools. At 5:00 a.m. he appeared listless and acutely ill, and developed generalized abdominal rigidity. No bowel sounds were heard, and he complained of severe generalized abdominal pain. The blood pressure was 120/75 and pulse 68 at this time. It was considered that he had perforated his upper gastrointestinal tract, but flat radiographs of the abdomen with the patient in the upright and horizontal positions failed to reveal free gas in the abdomen. It was suggested by the surgical consultant that supportive measures be continued and his progress observed.

By 10:00 a.m., March 5, his temperature had risen to 100.6° F., but his abdominal rigidity had relaxed, the abdominal pain was less, and a few faint bowel sounds were audible. His urine became a little lighter in colour, but still contained red cells, a few casts and a trace of albumin. He was placed on Dicrysticin, and supportive therapy continued. Hæmoglobin value at this time was 14.7 g., white cell count 13,500, and urea nitrogen 29 mg. %.

On the evening of March 5, he looked much improved and all abdominal pain had disappeared. He stopped having loose bloody stools, the urine cleared in colour and pathological content, and the abdomen was soft, although still tender in the epigastrium.

The patient was discharged on March 14. He made an uneventful recovery, showing no sign of oliguria, anuria or uræmia. His urine returned to normal in volume and quality, temperature fell to normal on March 7, stools were formed with no occult blood, urea nitrogen was 18.8 mg. % on discharge, and he tolerated a full diet.

The blood urea nitrogen on March 22, when he returned as an outpatient, was 22.4 mg. %.

DISCUSSION

It has been shown that the damaging effects of both arsenic and mercury depend upon their attachment to the sulfhydryl group of cellular proteins, and interference with the function of these essential cellular enzymes. If BAL is given before irreparable cellular damage has been done, it extracts the arsenic and mercury from the sulfhydryl group, and forms a harmless, readily soluble substance easily excreted in the urine.

Without BAL (dimercaprol) the ingestion of one tablet (0.5 g.) of mercury bichloride is usually not fatal, two tablets are fatal in 50% of cases, and more than two are usually lethal, invariably causing severe nephrosis, uræmia, and enteritis. It is especially noteworthy that, to be effective, BAL therapy should be given as promptly as possible, as little benefit is observed if it is given more than six hours after ingestion of the mercury. Vomiting within half an hour of ingestion probably prevents most absorption. The optimal gastric lavage fluid is sodium formaldehyde sulfoxylate, which combines with mercury; if this is not available, concentrated solution of sodium bicarbonate is a good second choice.

The recommended initial dose of BAL is 5 mg. per kg. body weight, with about half this dose in two hours, then repeated every four hours for about four doses, and slowly tapered

off over the next 36 to 48 hours. There are no clinical contraindications to the use of this drug, and the side-effects when seen are reported to be minimal and in no way dangerous. The only mistake that can be made in these cases of mercury bichloride poisoning is not to give enough BAL and to wait too long before instituting therapy. All doses should be given deeply intramuscularly to avoid sloughing of subcutaneous tissues.

The only large series of cases of mercury bichloride poisoning comes from Johns Hopkins Hospital, Baltimore, reported by Dr. W. T. Longcope.[2] He collected 61 cases over a period of three years, in which two patients died, one from inadequate doses of BAL given too late, and the other because she reached the hospital in a moribund condition many hours after swallowing the poison. The author was impressed with the rapid recovery of very seriously ill patients and the total lack of complications, as seen in this case. The effectiveness of treatment in all his cases depended not on the dose of mercury bichloride taken (which varied between 0.5 and 20.0 g.) but on the promptness with which BAL was given after ingestion of the poison. By reviewing earlier cases Longcope demonstrated that in cases treated by older approved methods, but without BAL, within four hours of ingestion of mercury bichloride, there was a 31.4% mortality; in those patients treated with BAL within four hours of ingestion of the poison there were no deaths and no complications.

It is of interest that mercury bichloride (commonly referred to as corrosive sublimate) can be obtained from any drug store by signing the poison registry, or by dairies directly from the manufacturer. Dairies apparently do not account for or lock up these tablets. Dairy tablets are round, sharp cornered and red speckled and contain 0.25 g. mercury bichloride. Tablets from drug stores are blue, coffin-shaped, and contain 0.5 g. Dairies use mercury bichloride to preserve milk samples for testing and it is easily accessible (as seen in this case). It can also be put in solution for gardening purposes to reduce fungus growth, and some use it for footbaths and douches.

SUMMARY

A case of mercury bichloride (corrosive sublimate) poisoning is reported. The patient ingested a lethal dose and vomited about one hour later, and BAL therapy was instituted within four hours of ingestion. The patient made an uneventful recovery with no complications or obvious kidney damage. There were no side-effects due to the BAL as given. The importance of giving adequate doses of BAL early in these cases is stressed.

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PRIMARY CARCINOMA OF THE DUODENUM

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Trail, B.C.

CARCINOMA of the duodenum is a relatively rare condition. In a survey of the literature, Eger[3] reported an incidence of primary carcinoma of the duodenum of 0.033% of 350,826 autopsies, and of 0.3% of all gastrointestinal carcinomata. In reviewing 228 cases of primary carcinoma of the small intestine, Hoffman and Pack,[4] however, drew attention to the fact that in 104 of these cases—that is, in 45.6%—the tumour was located in the duodenum. Considering the length of the duodenum and comparing it with the added lengths of the ileum and jejunum, it is clear that primary carcinoma of the small intestine shows a striking predilection for the former.

Carcinoma of the duodenum has been classified into suprapapillary, peripapillary and infrapapillary types, according to the relationship of the site of the tumour to the duodenal papilla. Kleinerman *et al.*[5] found their respective distribution in 453 cases to be as follows: suprapapillary, 102 (22.5%); peripapillary, 268 (59.2%); infrapapillary, 83 (18.3%). Much discussion exists as to whether peripapillary carcinomata do in fact arise from the duodenum, for at this site they could conceivably arise also from the ampulla of Vater, the pancreatic duct, the lower end of the common bile duct, or the mucosa of the papilla. Undoubtedly peripapillary carcinoma can and does arise from any one of these structures, and even the most careful histological examination may not lead to the

correct cytological diagnosis. Thus in a report on 229 ampullo-papillary growths, Lieber, Stewart and Lund[7] could only assign a definite origin to 14 cases (5.5%), as follows:

	Cases
Primary carcinoma of the ampulla of Vater	3
Primary carcinoma of the duct of Wirsung	1
Primary carcinoma of the lower end of the common bile duct	7
Primary carcinoma of the mucosa of the papilla	3
Growths involving structures 1-4	182
Growths involving structures 1-3	33

In the vast majority of instances the tumour is a columnar cell adenocarcinoma, although squamous metaplasia has been reported by Lieber, Stewart and Morgan.[8] Lymphatic involvement would seem to be much more common in the suprapapillary than in the peripapillary type. Thus Stewart and Lieber[12] reported glandular metastases in 75% of cases in the former instance, whereas d'Offay in 1946 reported only a 25% incidence in the case of the latter. In a recent report of five cases by Lunn[9] no lymphatic metastases were found in four cases, while in the fifth no lymph nodes were detected in the specimen.

The outstanding symptom is intermittent vomiting; the presence or absence of bile in the vomitus depends upon the relationship of the lesion to the duodenal papilla. Pain in the right hypochondrium passing to the back is a common complaint; anorexia and loss of weight follow soon after. Jaundice is often present in peripapillary lesions but is not invariable, as demonstrated in the case reported below. A mass may or may not be palpable; where jaundice is the case, Courvoisier's law may be exemplified by the finding of a palpable gallbladder.

Investigations are frequently negative, with the exception of the finding of occult blood in the stools, provided that repeated examinations are made. Duodenal aspiration may be of assistance by revealing the presence of blood and/or cancer cells; more often no abnormality is detected in the aspirate. Careful scrutiny of the duodenal loop in a barium series may lead to the diagnosis. Rogers, Goligher and Williams[11] have shown the value of performing the test during bouts of obstruction and vomiting; previously negative findings were then contradicted by demonstration of the site of obstruction. Not infrequently, however, a definite diagnosis can be made only at laparotomy.

Treatment consists in the adequate excision of the lesion, provided that it is still within resectable confines. Segmental resection with end-to-end duodeno-jejunal anastomosis is possible in some instances of carcinoma of the third part.[2] In peripapillary and suprapapillary carcinoma, pancreatico-duodenectomy, preferably in one stage, is to be recommended. Local transduodenal excision has been performed in the past, with relief in some instances, but the operation contravenes the time-proven tenets of sound cancer surgery. Gastro-jejunostomy and cholecyst-jejunostomy are valuable palliative procedures in those cases where the disease is too widespread for adequate resection. It is also recommended as a primary step in treating the debilitated patient not deemed sufficiently strong to withstand the more prolonged radical procedure in one stage. Subsequent improvement in such an instance may permit the performance of pancreatico-duodenectomy at a later date, although the presence of adhesions and of increased vascularity in the parts to be mobilized may well render the operation much more difficult than if it were being undertaken *ab initio*.

The prognosis is poor, regardless of the treatment. Survival beyond five years has, however, been reported by Eger[3] (one case), Berger and Koppelman[1] (4 cases), and Morley.[10] The relief afforded by the immediately successful operation is great, and a worthwhile extension of life is frequently obtained. Increased awareness of the condition and the bold attitude now being taken in its eradication lead one to hope for more successful results in the future.

Mrs. B.G.O., white, 65 years of age, first came under the attention of the surgical department in November 1953. She was complaining of intermittent lower abdominal pain and constipation. The previous history was not significant, except that a cholecystectomy had been performed many years previously, while she was residing in India, for cholelithiasis. On examination no definite abnormality could be detected. A barium enema had revealed the presence of diverticula in the pelvic colon. A single examination of the stools for occult blood was negative. A diagnosis of pelvic diverticulitis was made and a trial of conservative measures was advised. These consisted in the administration of a low residue diet, mineral oil by mouth daily, and a weekly enema. The importance of continued medical supervision was stressed.

The patient was seen again in January 1954 when she declared that the treatment "worked like a charm" and that she was symptom-free. Shortly afterwards she developed further pain which persisted, and she consulted her physician again late in March 1954. The pain had shifted to the right side of the abdomen opposite the umbilicus. There was severe nausea and a marked decrease in appetite. She remarked that an egg-nog relieved symptoms for two to three hours. Since

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November 1953 she had lost 10 lb. in weight. Bowel function was quite normal and regular. On examination there was some tenderness in both iliac fossæ, more marked on the right side. The urine was negative and still no occult blood was detected in a single stool specimen. A barium series was performed and no abnormality was reported. It was thought at the time that the patient was upset by the regimen prescribed for her diverticulitis, although tenderness persisted in the right side of the abdomen.

In April 1954 the patient complained that the pains were more severe and that there was complete loss of appetite. She had vomited the night before her visit to the Clinic and had brought up a large quantity of yellowish fluid, of gruel-like consistency. There was a further loss of 2 lb. in weight. Bowel function was still regular. On examination of the abdomen, tenderness persisted in both iliac fossæ; the edge of the liver, which was thin and smooth, could be palpated two fingerbreadths below the costal margin.

In view of the unsatisfactory progress the patient was admitted to hospital under the care of her physician. Pain now became more prominent and was epigastric. It was relieved by vomiting at frequent intervals of 12-14 ounces of bile-stained fluid. Weakness was now a prominent feature. The patient had lost weight and she was somewhat dehydrated. There was no jaundice. Marked tenderness and guarding were detected in the epigastrium. Results of laboratory investigations were as follows: red cells 5,070,000, white cells 7,150 (eosinophils 2, segmented 69, lymphocytes 23, monocytes 6); hæmoglobin value 96%; sedimentation rate 19 mm. in one hour. Urine negative. Blood cholesterol 150 mg. %, alkaline phosphatase 2.4 B.U., thymol turbidity 0.3%, colloidal gold flocculation +1, icteric index 7.6, plasma proteins 6.2 g. % (albumin 3.7, globulin 2.5). The bromsulphthalein test showed 55% retention of dye after 30 minutes. Stools were faintly positive for occult blood. An intravenous pyelogram was reported as normal.

The patient was seen in consultation on April 19 and the impression derived was that pelvic diverticulitis no longer accounted for the symptoms present and that some other lesion was at fault. This was thought to be in the nature of a duodenal obstruction; adhesions, chronic pancreatitis and carcinoma of the pancreas or ampulla of Vater were considered as the likeliest choices. Laparotomy was advised and accepted.

On April 29, abdominal exploration was performed through an extensive upper paramedian incision on the right side. Numerous adhesions were present between the liver, stomach, duodenum and transverse colon and the anterior abdominal wall, no other definite abnormality being then noted. When the adhesions had been sectioned and order restored, a vague mass became palpable in the right loin, not unlike a mass of calcified tuberculous lymph nodes. Further scrutiny showed the mass to correspond to the middle portion of the second part of the duodenum, its appearance and consistency being characteristic of carcinoma. There were no obvious secondaries either in the liver or the regional lymph nodes. A small, typical, benign ulcer was also present in the first part of the duodenum in a juxtapyloric position. The common bile duct was not distended. Trial dissection demonstrated that the inferior vena cava, the portal vein and the superior mesenteric vessels were free from carcinoma. A Whipple type of operation was then performed, the pyloric antrum, the whole of the duodenum, the head and neck of the pancreas and the proximal three inches of the jejunum being excised en bloc. Continuity of the alimentary canal was restored by end-to-side choledocho-jejunostomy, end-to-side pancreatico-jejunostomy (a small rubber tube having been threaded into the pancreatic duct) and end-to-side gastrojejunostomy. The wound was closed around a Penrose drain leading out from the region of the first anastomosis. The specimen removed is depicted in Figs. 1 and 2; the probe lies within the common bile duct.

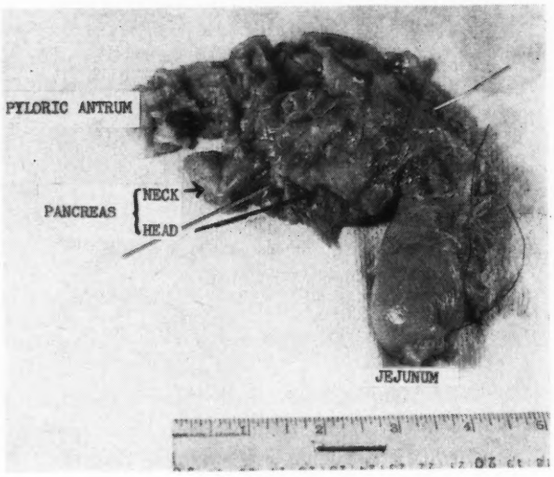

Fig. 1

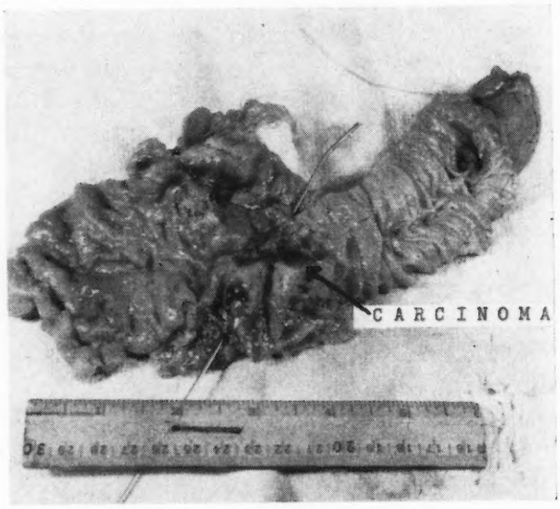

Fig. 2

Postoperatively, the patient developed a small biliary fistula which yielded readily to intermittent external aspiration; otherwise recovery was uneventful. Analyses of the stool on May 12 and 20, 1954, showed that both trypsin and amylase were present on each occasion; bile was present throughout. The patient was discharged on May 22, one month after operation, eating well and walking about freely.

PATHOLOGICAL REPORT

(a) *Gross description.*—32 cm. of resected small bowel incorporating the first, second, and third parts of the duodenum, some of the attached mesentery, and the pancreas. The organ has been opened along the lesser curvature to expose a large, circumferentially placed, constricting tumour at the mid-point of the second portion of the duodenum as it courses over the pancreatic head. This growth involves 4.5 cm. of the small bowel mucosa in a longitudinal direction. The mucosa overlying the tumour appears granular but no actual ulcer area can be accurately identified in the fixed state. The growth is both polypoid and fungating in type and does not appear to be ulcerated. On section, it presents a very dense, white, retracted cut surface and is continuous with the interstitial tissue of the pancreas. It is impossible to identify the ampulla of Vater, which may be involved in the tumour process.

Approximately 2 cm. from the site of proximal excision is a small ulcer area 0.6 cm. in diameter with rolled margins and punched-out appearance with adjacent thickened walls, but grossly not apparently malignant.

Occasional small lymph nodes are identified in the portions of attached mesentery.

(b) *Microscopic.*—Sections through the growths in the duodenum to include the underlying pancreas show in the latter no evidence of any carcinomatous process, but the duodenal mucosa is seen to be quite extensively eroded. In some places there is definite necrosis, and throughout the underlying duodenal wall there is a diffuse growth of very pleomorphic and polymorphic hyperchromatic epithelial cells apparently of columnar origin, many of them rather polyhedral showing many mitotic figures and generally large, deeply staining irregular nuclei; only in a very occasional instance is an extremely abortive attempt at acinar formation noted. The growth extends in some instances into the deeper layers of the muscularis but does not appear to extend beyond the duodenal wall.

Sections were taken through several of the accompanying lymph nodes but these do not appear to be involved by carcinomatous growth.

Sections taken through the small, punched-out, ulcer-like area mentioned above show loss of the mucosa over this site by ulceration and replacement by a semi-organized sanguino-fibrinous material, richly infiltrated with inflammatory cells, but there is no evidence of any malignancy at this site.

Diagnosis.—Anaplastic carcinoma of the duodenum.

Postoperative progress.—The patient was initially given small amounts of magnesium sulphate and olive oil by mouth every morning to assist the flow of bile into the small intestine. This was discontinued two months after discharge from hospital. Since then the stools have been somewhat paler than normal and therefore Entozyme capsules (250 mg. pepsin, 300 mg. pancreatin, U.S.P., and 150 mg. bile salts), one three times a day, have been prescribed. At the present time the patient is well and symptom-free and is leading a full, active and enjoyable life. Her only complaint is her failure to gain weight as fast as she would like to. Her present weight is 107 lb. compared with 121 lb. preoperatively; she is, however, gaining a little all the time. Recently she gave a party for her closest friends to celebrate the first anniversary of her operation.

I wish to express my thanks to Dr. D. J. M. Crawford for referring this case to me and for affording me the benefit of his long, wise experience, and to my colleague Dr. J. Stefanelli for his able assistance in the operating room and in the wards. To Dr. H. H. Pitts of Saint Paul's Hospital, Vancouver, B.C., I acknowledge my indebtedness for his pathological report and for the help he has given me so unstintingly in the present case, as in numerous other instances, before and since.

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INFILTRATING LOBULAR CARCINOMA OF BREAST

P. N. KARNAUCHOW, M.D.,* *Ottawa*

LOBULAR CARCINOMA *in situ* is recognized by many pathologists as a rare type of mammary cancer. Short descriptions of it are given in some textbooks of surgical pathology,[1, 2] and it is described and illustrated fully by Stewart in Section IX, Fascicle 34, of the *Atlas of Tumor Pathology,* published by the Armed Forces Institute of Pathology, Washington, D.C. Nevertheless this neoplasm, particularly in its infiltrative phase, has attracted comparatively little attention in the medical literature since its characteristics were clearly set forth by Foote and Stewart[3] in 1941. It is my purpose to give an account of a case of infiltrating carcinoma of the breast, believed to be of lobular origin.

A 33-year-old married white woman became aware of a lump in the left breast early in 1955. The lump was said to have increased in size during the latter part of 1954, causing her to seek medical attention. This was in another city, and information as to the findings on physical examination is scanty. Apparently the mass was considered to be a fibroadenoma, and in January 1955 a simple mastectomy was done. The surgical specimen was sent to this department for pathological examination.

PATHOLOGICAL EXAMINATION

Gross description.—The specimen consisted of a simply amputated female breast, measuring 12.0 x 8.0 x 5.0 cm., covered by an ellipse of freely movable skin, bearing a normal nipple. Sectioning showed adipose tissue surrounding an irregular mass of whitish-yellow, moderately firm, glistening tissue, measuring approximately 8.0 x 6.0 x 3.0 cm. This was studded with innumerable hard, pale greyish nodules, measuring up to 0.5 cm. in diameter (Fig. 1). The nodules were more numerous in the central and posterior portion of the specimen, where they showed some tendency to coalesce. The larger mammary ducts below the nipple showed moderate dilatation and contained yellowish-grey, paste-like material.

Microscopic description.—Numerous sections from representative sites in the breast were of essentially similar appearance. Small lobular areas, of the configuration of normal mammary lobules but somewhat larger, were composed of carcinoma cells which were usually arranged in a loose, haphazard fashion but in some instances formed short cords and occasionally gave a suggestion of acinar formation. The majority possessed large, pale nuclei and a large amount of pale, sometimes vacuolated, acidophilic cytoplasm. The nuclear-cytoplasmic ratio was little changed and mitoses were rare. Tumour cells of a somewhat different appearance were also seen. These varied considerably in size and shape, had intensely hyperchromatic nuclei and gave positive results when stained by the methods of Masson[4] and Lendrum[5] for demonstration of myoepithelium.

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In some areas the small lobular condensations of tumour cells were sharply outlined, but elsewhere the carcinoma cells extended into adjacent fibrous and fatty tissue. The invasive cells were predominantly myoid in type and very numerous, teeming singly, in small groups and in thin files in the dense fibrous tissue. Many small lymphatic channels were plugged with tumour cells. A few non-neoplastic lobules remained, some showing mild hyperplastic change. Others showed encroachment of invasive tumour from the periphery. A few lobules were the site of *in situ* carcinomatous change. The architecture of these was still intact but the component epithelial elements showed carcinomatous transformation, resembling cytologically the tumour cells in areas of frank invasion.

The larger ducts were moderately distended, some of them containing cellular debris and the others being empty. Their epithelial lining showed no neoplastic change. A few small ductules were lined by malignant cells. There was some periductal lymphatic infiltration.

COMMENT

The majority of primary carcinomas of the human female breast are believed to arise from the mammary ducts. Ewing,[6] while subscribing to this view, nevertheless recognized the existence of primary acinar carcinoma, referring to the form described by Cornil,[7] in which there was extensive multiplication of acini in rather well-defined lobules. Foote and Stewart in 1941 proposed the term lobular carcinoma for this neoplasm, choosing this term because of the general lack of agreement on whether acini exist in lobules of the resting breast. They described an *in situ* and an infiltrating phase, pointing out the distinctive microscopic pattern of each and stressing the likelihood of multicentric origin. In this regard it is interesting to note that in the monograph of Cheatle and Cutler[8] several photomicrographs (157 to 160), depicting two cases which they term "carcinoma arising in the epithelium of the entire gland or nearly the entire gland", suggest lobular carcinoma. In the medical literature since 1941, I have been able to discover only one paper dealing with lobular carcinoma of the breast, namely that of Godwin,[9] who, in 1952, recorded a case of infiltrating lobular carcinoma and made reference to two other cases of the *in situ* type.

The tumour in the case presented here did not resemble either grossly or microscopically the common varieties of carcinoma arising from lactiferous ducts. It was considered to be of lobular origin on the basis of the presence of lobular formations of loosely and haphazardly arranged tumour cells in association with a few mammary lobules showing *in situ* carcinomatous change. The pattern of invasion, consisting of single cells and small groups and threads of

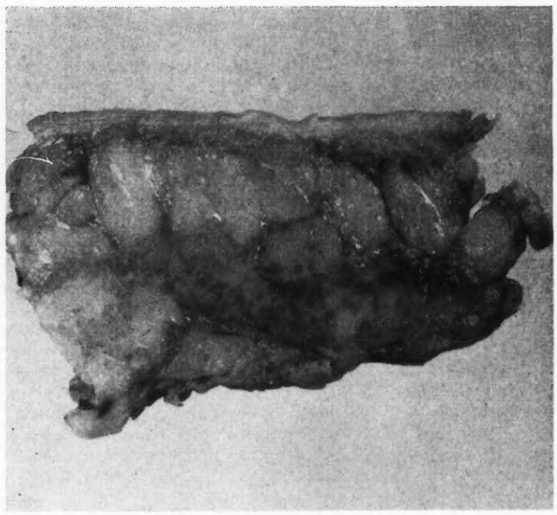

Fig. 1.—Infiltrating lobular carcinoma of breast. Cross-section of breast near nipple, surface-stained with hæmatin, to emphasize the diffuse distribution of greyish nodular masses of tumour tissue.

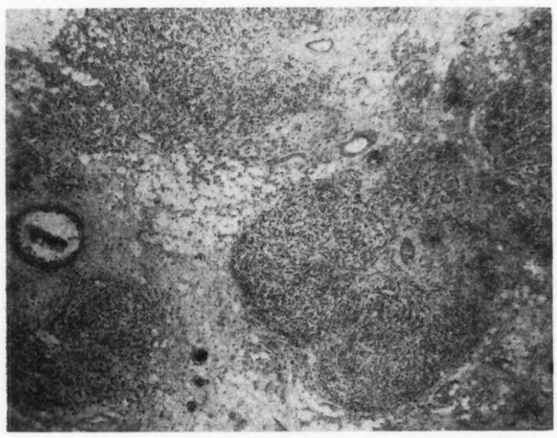

Fig. 2.—Infiltrating lobular carcinoma of breast. Masses of tumour cells still partially retain a lobular outline and tend to coalesce. Diffuse infiltration is seen at the upper left. At the left centre is a small duct containing cellular debris. Hæmatoxylin-erythrosin-saffron. × 36.

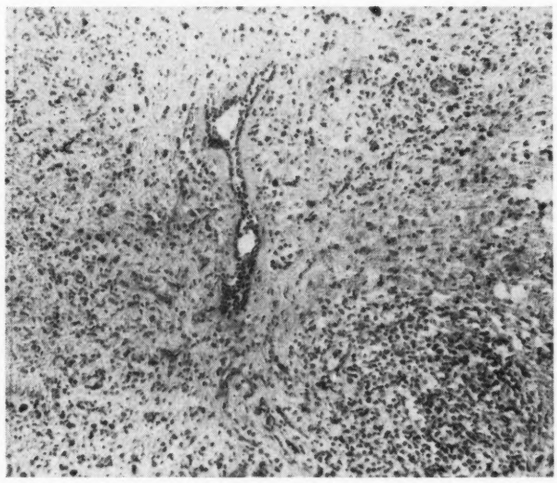

Fig. 3.—Infiltrating lobular carcinoma of breast. There is extensive diffuse infiltration of interlobular fibrous tissue by tumour cells, predominantly of the myoid type. At the lower right is a portion of a neoplastic lobule. Celestin blue-hæmalum-lissamine-tartrazine. × 120.

cells spreading diffusely in dense fibrous tissue, is not specific but is encountered infrequently in other forms of mammary cancer. The majority of the invading cells possessed the morphological and tinctorial characteristics of myoepithelium. This finding, also noted by Foote and Stewart, may represent myoid metaplasia of neoplastic epithelium. The presence of neoplastic cells within the terminal ductules, which may be interpreted as being due to tumour invasion from lobular areas or as representing a primary neoplastic change, has also been reported previously in association with lobular carcinoma.

According to Foote and Stewart, lobular carcinoma is of multicentric origin and progresses from the *in situ* stage to the phase of infiltration and metastasis in the space of a few months. However, in Godwin's first case, lobular carcinoma *in situ* was present in biopsied tissue 12 years previous to the development of clinically evident invasive carcinoma. Foote and Stewart believed that a simple mastectomy should be done when a rapid section reveals the presence of lobular carcinoma *in situ*, further procedures to depend on whether there is evidence of invasion on subsequent detailed pathological examination. However, since serial sectioning of the entire breast is not practicable, pathological evidence of non-invasiveness of an *in situ* growth must be considered as only presumptive, even if multiple sections from many carefully chosen blocks are examined.

SUMMARY

A case is presented of infiltrating carcinoma of the breast, believed to be of lobular origin.

I am indebted to Dr. D. Magner for criticism and help in the preparation of this paper.

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A CASE OF UTERINE HAMARTOMA

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HAMARTOMA is a relatively rare tumour, the name alone sending most of us to our dictionary rather than our case records. This tumour occurs most commonly in lung or mediastinum; unusual location in the uterus prompts the presentation of this case.

The patient, R.K., a married woman, aged 45, was seen in December 1954, suffering from a gastroenteritis. During examination a mid lower abdominal mass was palpated. This mass, about the size and shape of a small inverted pear, arose from the pelvis to about two inches (5 cm.) above the pubic symphysis and lay slightly to the right. It was smooth in outline, non-tender, and mobile in all directions but superiorly. Careful examination gave the impression that the mass was being held up by a larger cystic mass, filling the posterior part of the pelvis. A vaginal examination confirmed the presence of the larger cystic pelvic mass and that the tumour was merely a normal uterus pushed forward and upward so that it lay against the anterior abdominal wall, the cervix being above and posterior to the pubis.

Functional inquiry was essentially negative. There was a history of childhood poliomyelitis with residual wasting of the left leg. The patient, a thin, healthy-looking woman, was para 4, all normal, and the pelvis was known to be normal after the last birth in 1948. Her periods have continued unchanged to the present. She had been aware of the mass for five months. The urinary, gastrointestinal, respiratory and cardiovascular systems functioned normally. Blood, blood pressure and urine were normal. A provisional diagnosis of incarcerated left ovarian cyst was made and the patient sent to hospital for operation.

Laparotomy was performed on December 28, 1954, under Vinethene and open ether, with a left paramedian incision. The firm tumour was found to be an apparently normal uterus lying on a large cystic mass filling the pelvis. The cystic mass stretched retroperitoneally from the fundus to the sacrum and into the left broad ligament.

Incision of the peritoneum revealed a thick-walled, greyish, rather lobulated cystic structure, which was readily shelled out of the retroperitoneal space and left broad ligament. There was a broad fibrous base arising from the lower posterior uterine wall, and the fibrous strands appeared to run deep into the uterus. The cyst was clamped and excised. When opened, this cyst was seen to contain a gelatinous, spongy stroma with a lacy network of fine fibrils arising from the base and branching through its substance. On account of the invasive appearance of the base, a total hysterectomy was then performed, with closure of the vagina and reconstruction of the pelvic floor. The appendix was removed and the abdomen closed.

The patient was returned to the ward and intravenous therapy was begun. She reacted promptly, was sitting on the side of the bed by 8:30 p.m. and standing by 10:30 the same night; voiding was spontaneous. Penicillin and streptomycin were administered, with pyridoxine for nausea and intravenous fluid to control fluid balance. By the third day the patient was walking unassisted. The clips were removed on the seventh day and the patient was discharged with the incision healing nicely. Her smooth convalescence continued at home.

The first *pathological report* dealt with the appendix and uterus: "The uterus measures 12 x 6.7 x 4 cm., the

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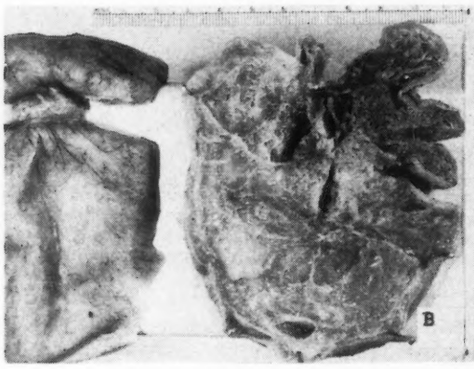

Fig. 1

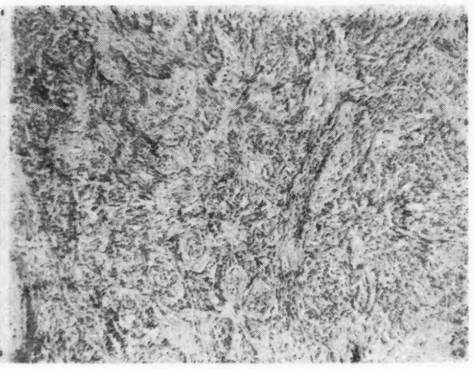

Fig. 2

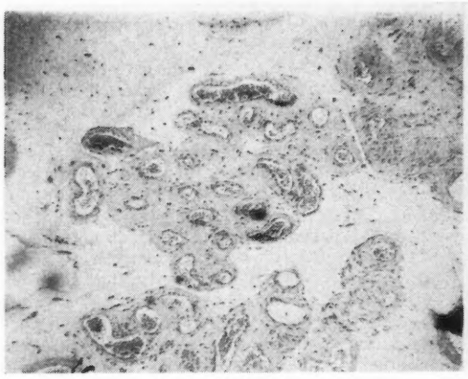

Fig. 3

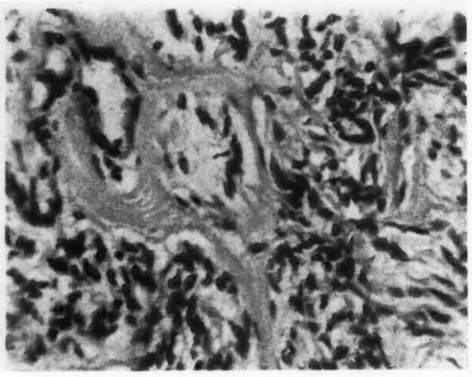

Fig. 4

myometrium 0.3 cm. in thickness, the endometrium 0.3 cm. in thickness. The latter is congested and polypoid in contour. On section the cervical tissue reveals cysts, which contain solidified mucoid material.

"The appendix measures 8 cm. in length by 0.6 cm. in diameter. Section shows obliteration of the lumen. The mucosa is intact. The submucosa shows slight thickening.

"The benign endometrium is well advanced in the secretory phase. The myometrium is somewhat fibrotic, consistent with the age of the patient. The cervix is covered in part by squamous cell epithelium, and the remainder by a single layer of tall columnar mucus-secreting cells. In the subepithelial and periglandular fibrous tissue, there is a mild infiltration of chronic inflammatory cells. A number of the mucus-secreting gland acini are cystically dilated. There is no evidence of malignancy. *Diagnosis:* secretory endometrium; chronic cystic cervicitis; hypertrophy of cervix; chronic fibrosing appendicitis."

On January 7, 1955, the pathologist reported:

"This lesion has given us some difficulty due to the fact that there is a great deal of degeneration throughout it. It is benign and appears to be either a vascular lesion or a degenerating fibromyoma. We have done special staining of the tissue, but it requires some further work before we can make a final diagnosis."

A final report (January 24, 1955) stated: "This fluctuant, oval mass is 9.0 cm. in diameter. Section reveals one-half the specimen to be formed of spongy, pink tissue with collections of moderately firm, granular, pale material. Adjacent to this is gelatinous, green material which is encapsulated.

"We have spent a good deal of time on this tumour from the surface of the uterus, and it has been seen by a large number of pathologists, including our consultant, Professor Hamilton, of the Banting Institute. It is a hamartoma. It is an anomalous development of the blood vessels, most of which are small arteries with moderately thick walls. The stroma shows myomatous change in many areas. The tumour appears benign histologically, except in the sections taken from the outer wall of the uterus. Here there is some activity, where the tumour is invading the outer part of the myometrium. Occasional lymphatics are also adjacent to the small vascular channels. This is a hamartoma of blood vessels, which is probably benign but, because of the area of activity, leaves some doubt in our minds. *Diagnosis:* hamartoma."

Fig. 1 shows the thick-walled cyst and the gelatinous fibrous appearance of its contents. Figs. 2 and 3 show the combination of fibrous tissue and bundles of relatively thick-walled vessels set in a loose myxomatous stroma. Fig. 4 shows the loose areolar structure, the thick-walled arteries, and the fibrous stranding of the tumour.

DISCUSSION

In review of the literature very little could be found concerning hamartoma in recent years. Albrecht describes these anomalies as error tumours, or overdevelopment of tissue that belongs normally in that site. Some authorities be-

lieve that they represent fetal arrests or displaced tissue. Hamartomas have been reported in kidney, liver, lung, mediastinum, and heart, but no recent mention of pelvic origin can be found. Cooper and Pecora[1] report two cases in lung, both of which contained cartilage, epithelial tissue, and loose fibrous and fatty tissue. A similar type in the mediastinum is reported by Boyd.[3] Hulse and Palik[2] report two renal hamartomas, both of which contained adipose tissue, fibrous tissue, and smooth muscle, with a tendency to form networks of arterioles and capillaries. They relate one renal hamartoma to tuberous sclerosis from which the patient was suffering, but in the other case a diagnosis of hamartoma was made without any evidence of tuberous sclerosis; from other literature, it would appear that the finding in the first case was coincidental. MacCallum[4] classifies hamartomas in the group of angiomatous tumours, while Price Thomas[5] classifies them as lung tumours solely, composed of cartilage, epithelial elements and muscle.

With the rather divergent opinion as to the etiology of this tumour, as well as the variation in its structure, it is not surprising that the present tissue caused some concern amongst the pathologists as to its correct identification. If the definition of Albrecht is true, one would expect in this case a vascular, fibrous type of tumour, arising as it did from the uterine wall. One would, however, hesitate simply to excise the mass and leave such a fertile bed of "roots" in the uterine wall, and the more radical procedure of total hysterectomy seemed justified.

SUMMARY

A case of uterine hamartoma is presented with a brief review of current literature.

Acknowledgment is hereby made to the Department of Health of Ontario for their diagnosis in this case and their courtesy in providing the photograph and photomicrographs.

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Special Article

A REPEAT SURVEY OF CANCER IN MIDDLESEX COUNTY, ONTARIO*

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A. H. SELLERS, B.A., M.D., D.P.H.,† *Toronto*

DEATH REGISTRATIONS give a little information about nearly all cases of fatal cancer: the age at death, the site of disease, sometimes its duration. Clinic records give a great deal of information about some cases of cancer. But there are many other cases treated elsewhere than at the Clinics, whose existence is known only to the treating physician, and perhaps not to the patient himself or to the physician who eventually signs the death certificate. If we do not know how many of these other patients there are and what becomes of them, we cannot really evaluate the treatment given at the Clinics; we cannot make well-founded plans for treatment facilities; and we cannot say whether the morbidity and fatality of the disease, as distinct from its mortality, are rising or falling.

METHOD OF STUDY

In 1940 a pilot survey of the incidence and prevalence of cancer was conducted, in retrospect for the year 1939, in Middlesex County, Ontario.[1] The survey findings have proved useful as an index of the amount of cancer in the Province; and in accordance with the recommendations made by the Survey Committee at that time a repeat study has been carried out in the same county for the year 1953. Since surveys of this kind are generally considered to provide the best available picture of the amount of cancer in a given area, the results may again prove interesting in spite of the relatively small numbers involved.

The 1953 Middlesex Survey was conducted under the joint auspices of the Ontario Medical Association, the Ontario Cancer Treatment and Research Foundation, the London Academy of Medicine, and the Department of Health for Ontario. In order that comparisons might be made with the earlier study, the County of Middlesex was again chosen as the survey area, and the study was again made in retrospect.

The County of Middlesex was estimated to have a population of 170,180 in the year 1953: 84,034 males and 86,146 females. Approximately 98,850 people reside in the City of London. The population of the county is slightly older than the general population of Ontario. The county is well provided with physicians and hospitals, and the London Clinic of the Ontario Cancer Foundation has excellent facilities for the treatment of cancer, including a cobalt-60 beam therapy unit.

Late in 1953 copies of the survey schedule were mailed to all physicians, hospitals and laboratories in Middlesex County, after advance notification. Follow-up letters and visits by the medical field-worker, a Middle-

*This article was prepared for the Sub-Committee on Cancer Surveys of the Ontario Cancer Treatment and Research Foundation. The project was supported by funds made available by the Foundation.
†From the Division of Medical Statistics, Department of Health for Ontario.

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sex physician, helped to bring in the replies. In addition, all death registrations with mention of cancer, all other Cancer Clinic new-case records, the Ontario Cancer Foundation Biopsy Service reports, and the monthly returns of all public general hospitals in Middlesex and the adjacent counties, were checked for Middlesex residents with cancer during the study year. Cards were made out for all reported cases, duplications were eliminated, and information was transcribed to dual-purpose IBM punch-cards for sorting by machine in the Division of Medical Statistics.

Of 345 physicians approached, 135 were interns, specialists in fields unrelated to cancer, or not in practice. Of the remainder, 68 reported that they had seen no cancer cases in 1953; 39 stated that they referred all their cases to hospital or the Clinic; 73 physicians made a total of 597 reports on 464 cases among Middlesex residents; and 30 physicians did not respond, resulting in an estimated under-reporting of perhaps 2% both for all new cases and for all cases with cancer.

TABLE I.

REPORTED CANCER INCIDENCE, PREVALENCE, TOTAL CASES AND MORTALITY
RESIDENTS OF MIDDLESEX COUNTY, 1953

Group	Number of cases			Rate per 100,000 estimated population			Ratio of cases to deaths
	Male	Female	Total	Male	Female	Total	
Incidence: (New cases: onset or first diagnosis in 1953)	210	249	459	250	289	270	1.6
Prevalence: (History of cancer; cancer not stated to be absent in 1953)	372	462	834	443	536	490	2.9
Total cases: (All cases with history of cancer)	599	755	1,354	713	877	796	4.7
Mortality: (Deaths from cancer)	137	151	288	163	175	169	—

VOLUME OF CASES REPORTED

For Middlesex residents, a total of 2,352 reports were received for 1,354 cases in 1,338 patients. Fourteen patients had second independent primary lesions, and one patient had a third lesion: in all tables in this report the term "case" refers to an independent primary lesion, and not to a patient.

Included among the reported cases were 193 patients apparently cancer-free five to 16 years after the microscopically confirmed diagnosis of cancer: the principal sites in this group of long-term survivors were skin (70 cases), breast (37 cases), buccal cavity and pharynx (31 cases), cervix uteri (26 cases), and corpus uteri (7 cases). No serious effort was made to search out cancer-free cases, and only those who were seen for follow-up or other reasons are reported.

INCIDENCE, PREVALENCE AND MORTALITY

The chief findings of the survey with regard to incidence, prevalence and mortality are summarized in Table I.

Based on the 1,354 reported cases of cancer among Middlesex residents, the following rates were obtained: incidence (new cases diagnosed during the year), male 250, and female 289 cases per 100,000 population of the same sex; prevalence (all cases with cancer during the year or not stated to be free of cancer), male 443, and female 536 cases per 100,000 population; mortality from cancer, male 163, and female 175 cases per 100,000 population. The ratio of new cases to deaths was 1.6 to 1, as in the 1939 survey. The ratio of all cases with cancer to deaths was 2.9 to 1, and of all cases with a history of cancer to deaths, 4.7 to 1. If skin cancer be excluded, these ratios become, respectively, 1.3 to 1, 2.5 to 1, and 3.6 to 1.

A total of 578 cases or 340 per 100,000 population were reported as receiving treatment (not necessarily for the destruction of the tumour) in 1953.

The crude incidence rates for both sexes, and the crude mortality rate for females, were similar to the crude rates observed for 1939. The crude prevalence rates for both sexes, for treated cases, were 21% higher than the 1939 rates. The crude mortality rate for males was 9% higher than the corresponding rate in 1939. For comparison, the Ontario cancer mortality rate for females was similar in 1939 and in 1953, though considerably lower than the Middlesex rate; and the Ontario cancer mortality rate for males increased by 18% from 1939 to 1953.

VERIFICATION OF DIAGNOSIS

Microscopic confirmation of the diagnosis of cancer was available for 82% of all cases reported in 1953, as compared with 63% of those reported in 1939. In 1953, 89% of cases reported by hospitals or the Cancer Clinic were confirmed. The increase in the proportion of confirmed cases was roughly the same for both accessible and inaccessible sites (29% increase for oral cavity, breast, genital organs and skin, and 33% increase for the remaining sites). A good deal of improvement may be credited to the Ontario Cancer Foundation Biopsy Service, since 188 cases not reported by hospital or Clinic are stated to have had microscopic confirmation.

EXTENT OF DISEASE

Of the 459 new cases reported in 1953, 55% had localized disease, 18% had regional involvement, and 22% distant metastases; these figures are based on the most advanced extent reported for each case during the year. The highest proportion of cases with distant metastases was found in cancer of the stomach (48%), then prostate (44%), digestive system other than stomach

TABLE II.

REPORTED CANCER INCIDENCE, PREVALENCE, AND MORTALITY
BY AGE GROUP AND SEX
RESIDENTS OF MIDDLESEX COUNTY, 1953

	Incidence *		*Prevalence* †		*Total cases* ‡		*Mortality* **	
	Male	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*
Age group	*Number of cases*						*Deaths*	
0 - 24 years	—	8	6	12	7	15	2	3
25 - 34 "	5	5	10	15	19	20	4	1
35 - 44 "	15	21	17	38	36	58	3	9
45 - 54 "	21	33	37	71	61	123	19	19
55 - 64 "	44	54	84	90	126	170	39	32
65 - 74 "	65	74	110	127	191	201	30	44
75 - 84 "	44	41	75	83	114	129	28	33
85 and over	12	10	21	21	29	27	12	10
Not stated	4	3	12	5	16	12	—	—
Total	210	249	372	462	599	755	137	151
Age group	*Rate per 100,000 population*							
0 - 24 years	—	††	††	36	††	45	††	††
25 - 34 "	††	††	72	104	137	138	††	††
35 - 44 "	126	179	143	323	302	493	††	††
45 - 54 "	222	349	391	752	645	1,302	201	201
55 - 64 "	571	692	1,091	1,154	1,637	2,179	507	410
65 - 74 "	1,213	1,234	2,053	2,118	3,564	3,353	560	734
75 - 84 "	1,970	1,449	3,357	3,934	5,103	4,560	1,253	1,166
85 and over	3,061	1,451	5,357	3,048	7,398	3,919	3,061	1,452
Total	250	289	443	536	713	877	163	175

*Cases first diagnosed in 1953.
†Cases with cancer present or not stated to be absent in 1953.
‡All cases with history of cancer, whether or not cancer was reported to be present in 1953.
**Deaths from cancer in 1953.
††Rates not calculated for fewer than 10 cases or deaths.

and intestine (35%), and intestine except rectum (31%). In cancer of the buccal cavity and pharynx, larynx, bladder, brain, bone, and skin, cancer was reported as localized in more than two-thirds of the reported cases for each site.

The extent of disease records in the 1939 and 1953 surveys cannot be compared, as the criteria were different in the two studies. Of the new cases registered at the Ontario Cancer Clinics in 1954, 62% were reported as localized, 25% as having regional involvement, and 10% as having distant metastases.

SITE, SEX, AND AGE VARIATIONS

The incidence, prevalence, total cases and mortality are shown by age group and sex in Table II, and by primary site of disease in Table III. The numbers of cases are small, and the rates must be regarded as approximate only.

From 1939 to 1953, the crude incidence rate for males showed a more than five-fold increase for respiratory cancer, a more moderate increase for urinary system cancer, and a rather sharp decrease for buccal and genital system cancer. The crude incidence rates for females showed a three-fold increase in cancer of the urinary system, and a 30% drop in the reported breast cancer rate. The numbers of cases in many categories are so small that more or less complete reporting on the part of a single specialist might account for considerable differences. The crude death rates do, however, appear to support the evidence of an increase in male respiratory cancer and a decrease in male buccal and genital cancer.

Both the incidence and the prevalence rates for males are considerably lower than the female rates at ages below 75 years, and considerably higher than the female rates in the oldest age groups. A similar difference in the age-specific rates was noticed in 1939.

SOURCE OF REPORTS

Of the new cases, 56% were reported by hospitals, 47% by physicians, 39% by laboratories, 36% by the Cancer Clinic, and 26% by death registration. Two per cent of the new cases and 10% of all cases with cancer were reported only by death registration; some of these patients may have died in hospitals outside Middlesex county.

The duplication of reports in the present survey was a source of concern to many physicians. That it was necessary in order to obtain anything like a complete picture is obvious from the fact that only the hospitals reported more than half of the total number of new cases. If the physicians had not been circularized, 12% of the new cases and 11% of all cases of cancer would have been overlooked; the loss in new cases

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TABLE III.

REPORTED CANCER INCIDENCE, PREVALENCE, AND MORTALITY
BY PRIMARY SITE AND SEX
RESIDENTS OF MIDDLESEX COUNTY, 1953

	Incidence *		*Prevalence* †		*Total cases* ‡		*Mortality* **	
	Male	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*
Primary site	*Number of cases*						*Deaths*	
Buccal cavity and pharynx	15	4	26	6	95	15	4	1
Stomach	21	8	31	15	32	17	22	6
Other digestive system	36	58	67	96	69	109	40	53
Lung and bronchus	16	4	28	6	28	6	18	4
Other respiratory system	7	1	10	2	15	3	1	1
Breast	1	47	1	116	1	211	—	34
Uterus	—	31	—	59	—	124	—	15
Prostate	16	—	47	—	47	—	17	—
Other genital system	2	16	4	24	9	28	—	8
Urinary system	22	17	38	26	44	33	9	7
Skin	50	36	77	59	207	139	6	3
Lymphatic and hæmatopoietic	12	7	29	20	29	27	12	9
Other sites	12	20	14	33	23	43	8	10
All sites	210	249	372	462	599	755	137	151
Primary site	*Rate per 100,000 population*							
Buccal cavity and pharynx	18	††	31	††	113	17	††	††
Stomach	25	††	37	17	38	20	26	††
Other digestive system	43	67	80	113	82	127	48	62
Lung and bronchus	19	††	33	††	33	††	21	††
Other respiratory system	††	††	12	††	18	††	††	††
Breast	††	55	††	135	††	245	—	39
Uterus	—	36	—	68	—	144	—	17
Prostate	19	—	56	—	56	—	20	—
Other genital system	††	19	††	28	††	32	—	††
Urinary system	26	20	45	30	52	38	††	††
Skin	60	42	92	68	247	161	††	††
Lymphatic and hæmatopoietic	14	††	34	23	35	31	14	††
Other sites	14	23	18	38	27	50	††	12
All sites	250	289	443	536	713	877	163	175

*Cases first diagnosed in 1953.
†Cases with cancer present or not stated to be absent in 1953.
‡All cases with history of cancer, whether or not cancer was reported to be present in 1953.
**Deaths from cancer in 1953.
††Rates not calculated for fewer than 10 cases or deaths.

would have been greatest for stomach (24%), prostate (19%), and respiratory and urinary systems (18% each).

DISCUSSION

Can the results of the Middlesex survey be applied to estimate the size of the cancer problem in Ontario as a whole? If the numbers of cases were larger, the Middlesex age-specific rates might be applied to the Ontario population data; but as it is, the best index is probably formed by the case-to-death ratios. Applying these to the 6,782 deaths from cancer among Ontario residents in 1953, we would expect that there were in the province some 11,000 newly diagnosed cases of cancer in that year, some 20,000 cases with cancer at some time during the year, and some 12,200 cases requiring follow-up after apparent arrest of the disease. If skin cancer be excluded, there would be about 9,000 new cases, 17,000 cases of cancer, and 7,500 cases requiring follow-up during the year. Rough as these estimates are, they are perhaps the best we have at the present time.

The 1953 Middlesex Survey, through the co-operation of physicians, medical librarians and records staff, and medical organizations, revealed much about cancer incidence and prevalence in this part of Canada. It provided useful information about the volume of cases seen by various groups, about the accuracy of diagnosis, and some of the trends of disease, all data helping us to see the cancer problem in better perspective.

The present report can only touch on the highlights; a copy of the full Survey Report may be secured from the Ontario Cancer Treatment and Research Foundation on request.

REFERENCE

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CLINICO-PATHOLOGICAL CONFERENCE. I.

MONTREAL GENERAL HOSPITAL, NOVEMBER 17, 1955

TERENCE C. TODD, M.D.,
H. ERNEST MacDERMOT, M.D. and
WILLIAM H. MATHEWS, M.D., *Editors*

*Dr. Terence C. Todd:**

I wish at this time to express appreciation to Dr. Benjamin Castleman, Pathologist-in-Chief of the Massachusetts General Hospital, and to his assistant Virginia W. Towne, for much valued help.

PROTOCOL

A 32-YEAR-OLD WHITE MALE of Italian extraction was admitted on January 15, 1949. His complaints were of weakness, loss of appetite, fullness, loss of weight, difficulty in breathing, pain in the right side of the chest, and slight cough.

The family history was non-contributory. The patient was born of Italian parents and lived in Montreal all his life. He was a taxi-driver by trade and had never had a dusty job. He was in the Army in World War II (1942-1945) and was discharged at the end because of claustrophobia and a nodule in the left side of the thyroid gland.

SUMMARY OF ADMISSION THREE MONTHS AGO

The patient was in good health until about August 1948, when he developed pain in his left lower limb from hip to mid-thigh. Physical and x-ray examinations during the ensuing two months failed to reveal any cause for the pain, which became increasingly worse. Patient was admitted to hospital on October 21, 1948 for investigation. At the same time he was suffering from epigastric distress, which was dull and constant but aggravated about one hour after taking food, and not relieved by any home remedies. There was no radiation of this pain, but there was an associated back pain and the leg pain described above. He also developed a non-productive cough. He had some frequency, D/N 8.9:3.4.

Physical examination.—He was a healthy-looking male weighing 174 lb., lying in bed in mild distress from abdominal pain. The left lobe of the thyroid was firmly enlarged, with mild pain on pressure. There was no lymphadenopathy, and the chest and cardiovascular system were not abnormal. The abdomen was tense; deep palpation was not possible in the epigastrium. The liver was two fingerbreadths below the costal margin by percussion. No masses were felt. There was a healed transverse appendectomy scar, and a firm, tender area in the lower pole of the right epididymis.

The admission diagnoses were: (1) duodenal ulcer; (2) pancreatitis; (3) cholecystitis. The illness ran an afebrile course and his pain was intermittent. A barium series revealed no evidence of disease.

Laboratory findings.—White cell count 12,500; urinalysis negative; urinary diastase 26,000 units/24 hours (8,000-30,000); blood urea nitrogen 14 mg. %; blood sugar 105 mg. %; bilirubin 0.2 mg. %.

On November 3, 1948, two weeks after admission, an exploratory laparotomy was done with a preoperative diagnosis of recurrent pancreatitis. No evidence of disease was found in the stomach, small or large bowel, spleen or kidneys. The gallbladder was greatly distended and infiltrated with fat. The common bile duct did not appear enlarged, but the gland at the neck of the gallbladder was definitely enlarged. The pancreas was thickened, firm and œdematous and of uniform consistency from head to tail. There were no areas of fat necrosis.

An operation was carried out. Bile under pressure escaped from the gallbladder and the mucosa showed definite cholesterolosis, but no stones were found. The patient's postoperative course was uneventful and he was discharged to his home 10 days later, but still complaining of some pain in the epigastrium and in the left leg. He was followed up outside privately, and one month postoperatively a chest radiograph showed evidence of parenchymal disease.

PRESENT ADMISSION

The patient was readmitted to Montreal General Hospital one month later, i.e. in January 1949, two months after operation. His complaints were as listed—weakness, loss of appetite, fullness, loss of weight, difficulty in breathing, pain in the right side of the chest, and slight cough.

Physical examination.—He was now thin and apprehensive, giving evidence of very considerable wasting and pallor and severe dyspnœa, and moderate cyanosis. There was smooth enlargement of the thyroid with one large nodule in the left lobe, firm in consistency and the size of a golf ball. Lymph nodes just behind both lobes of the thyroid were moderately enlarged and shotty, and shotty and tender nodes were felt in both axillæ. There were moist rales in both lungs, and no C.V.S. abnormality. B.P. 110/80. There was upper abdominal fullness and lower abdominal retraction, with epigastric tenderness, a palpable liver, a questionably palpable spleen, a palpable right kidney, bilateral costo-lumbar tenderness, and a palpable nodule in the scrotum.

Laboratory findings.—Urinalysis showed occasional red cells. The hæmogram was as follows: R.B.C. 5,140,000; W.B.C. 21,300; Hb 102%; cell diameter 7.5; colour index 1.0; differential—polymorphs 88%, lymphocytes 6%, eosinophils 4%, metas 2%. Marked shift to the left. Some variation in size, shape and staining of erythrocytes. Blood chemistry: urea nitrogen 15 mg. % (10-20); sugar (fasting) 158 (80-20); bilirubin 0.1 (0.1-0.2); phosphatase—acid 3 units (0.5-4.0), alkaline 8 units (3-13); cholesterol 171 (150-200). Twenty-four-hour urine specimens (2), gastric washings (2), and sternal marrow smear were negative for tuberculosis. Sternal marrow sections showed no evidence of tuberculosis or other disease.

X-RAY EXAMINATIONS

1. *Chest film January 15, 1949.*—"The heart is normal in size, shape and position. The aorta is not widened. The trachea is in the midline. The hilar shadows are moderately prominent. These appear to be the branches of the pulmonary artery, rather than enlarged hilar nodes. There is evidence of diffuse and advanced disease involving the parenchyma of both lungs. The lesions are nodular in appearance but in the right upper lobe the lesion is confluent, producing a bronchopneumonia type of distribution. The nodules are not of equal size, and this tendency to confluence is some evidence against miliary tuberculosis. The picture is, however, consistent with the diagnosis of miliary tuberculosis, but on the basis of the distribution we would suggest that it is more likely due to pneumoconiosis or other type of interstitial lesion of the lung."

2. *Flat plate of the abdomen.*—No abnormality except the lung bases.

3. *Intravenous pyelogram.*—No definite abnormality.

COURSE IN HOSPITAL

This 32-year-old white man was admitted to the surgical service and was seen frequently in consultation by the medical service. A urologist considered that there

*Clinical Assistant, Medicine, Montreal General Hospital.

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was bilateral epididymitis, chronic in nature and almost certainly tuberculous.

The patient ran a low-grade fever of 99-100° F. throughout, and apparently had been febrile at home before admission. His course in hospital, a total of nine days, was steadily and rapidly downhill, showing deterioration daily. He was transferred to the medical service for management and streptomycin therapy. On the day of transfer he began to show some evidence of meningeal irritation, and a lumbar puncture was done. The cerebrospinal-fluid pressure was not elevated, and tests showed no abnormality. A short while later the patient expired, with no final dramatic episode other than elevation of temperature to 101° F.

DIFFERENTIAL DIAGNOSIS

*Dr. Edward S. Mills**

This case concerns an Italian male of 32, a taxi-driver; the total duration of the illness of this patient was about seven to eight months. The history includes symptoms pointing to the respiratory system, the upper abdomen and the left thigh.

On examination the following findings were elicited: a nodule in the epididymis; enlargement of the left lobe of the thyroid; a tense upper abdomen which could not be properly examined because of tenderness and tenseness; and finally, a leukocytosis.

After a considerable amount of study on the surgical wards, his abdomen was explored, with preoperative diagnosis of recurrent pancreatitis. We are not told what operation was performed, but assume that the gallbladder was removed. When it was examined from the inside it showed cholesterolosis. It was noted also that the pancreas was indurated throughout its entire length, and there was bile under pressure in the gallbladder. These facts might indicate that there was disease of the pancreas. We are told that there was no evidence of disease of the stomach, small bowel, kidneys or duodenum. At this time no evidence of disease of the lungs was found.

After operation he continued to have pain in the left thigh and the same type of pain in the upper abdomen. After a month or two a chest radiograph showed widespread disease of the lungs. He went rapidly downhill, and within three months had died, having developed marked cachexia, fever and extensive bilateral disease of the lungs. The interesting feature is the rapidity with which he became cachectic and emaciated.

I would like to show the x-ray appearance of the lungs. Unfortunately we have no record of the radiograph taken a month after his operation. The one now shown was taken during his final admission, and not very long before his death.

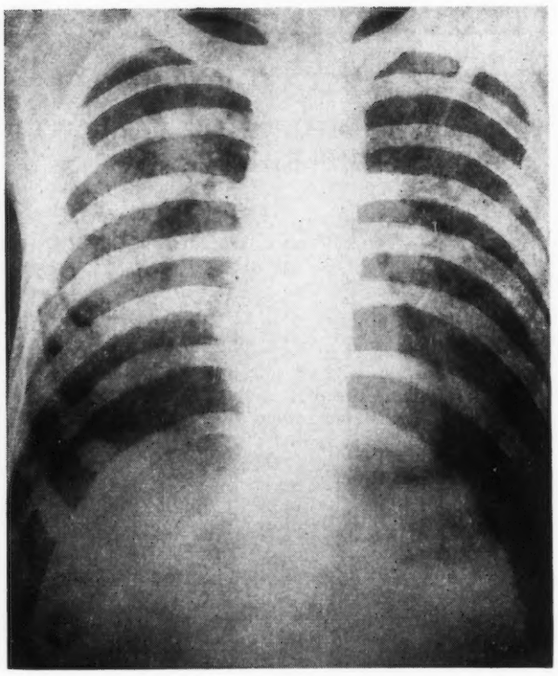

Fig. 1

The radiograph shows widespread involvement of both lung fields, with some confluence of the lesions on the right side, as described in the protocol. The heart appears normal. I asked Dr. McKay* if he had any comments to make on this film, and he told me the appearances could be due to any one of 22 different diseases.

We now come to the question of the differential diagnoses. I am going to consider first the things I consider less likely:

1. BOECK'S SARCOID
2. HISTOPLASMOSIS OR FUNGOID DISEASE OF THE LUNG

There are a number of reasons why these diagnoses seem unlikely. First of all, the rapidly developing course is very much against either of them. It is true that Boeck's sarcoid can and does affect a number of systems such as the abdomen and lungs, with bouts of peripheral neuritis, but neither of these diseases kills within a period of seven months. I therefore think it unlikely that we are dealing with either Boeck's sarcoid or one of the fungoid diseases of the lung.

*Physician-in-Chief, Montreal General Hospital.

*Dr. J. W. McKay, Radiologist-in-Chief, Montreal General Hospital.

3. CARCINOMA OF THE THYROID

You will have noted that there was an enlargement of the left lobe of the thyroid—again found at the time of the first admission—and at the time of the discovery it was described as "a nodule as large as a golf ball". Also glands were noted "behind the thyroid". I am not sure how one could recognize these, but nevertheless they were found. So one might also bear in mind carcinoma of the thyroid, and this is likely to metastasize to bones and might produce a root pain, but we have no evidence either clinically or radiologically that there are any metastases, and this diagnosis would not explain the distended gallbladder and the œdematous abdomen. So I think one can perhaps assume that the nodule in the thyroid was not due to primary carcinoma in the thyroid gland.

Another finding recorded was a nodule in the epididymis. If the nodule was in the testis, one might consider the possibility of carcinoma of the testis. However, at the time of the second admission it was discovered to be bilateral, and the urological consultant indicated that it was almost certainly tuberculous. So that I think one can exclude the nodule in the epididymis as the primary site of tumour.

4. HODGKIN'S DISEASE

This was a young man, 32 years of age. He had no fever to start with, but did develop one as time progressed. He had an enlarged gland at the neck of the gallbladder, and some disease process in the upper abdomen which we are told was not in the stomach, duodenum, small bowel or kidneys, and he had eventually some questionable enlargement of the spleen. There were a few shotty glands noted towards the end of his illness. So one might possibly consider Hodgkin's disease. But I am inclined to discount this diagnosis for several reasons. One is that Hodgkin's disease does not as a rule spread in this way throughout the lung. Dr. McKay has informed me that he considers the spread throughout this lung was likely to be a lymphatic spread, and I am not so sure that such a lesion could be due to Hodgkin's disease. Then again, the course was rapid. The course of Hodgkin's disease is anywhere from 2 to 25 years, and invariably before the patient dies there is a general adenopathy. We are told the spleen was questionably involved. One cannot of course decide whether it was or not, and there was some doubt as to whether there was generalized involvement of the lymphatic system. The fever was of low-grade type, not having the characteristic Murchison or Pel-Ebstein variation which we expect in Hodgkin's disease; I therefore think that one can probably exclude Hodgkin's disease as a cause of this man's illness.

5. TUBERCULOSIS

There is a possibility that the lesion in the lung was tuberculosis. He had what is considered by the urologists to be tuberculosis of the epididymis. Is it reasonable to assume that the lesion of the lung is tuberculous? Cultures, bone marrow specimens and gastric washings did not reveal the tubercle bacillus.

You will note that towards the end of the protocol it is stated that after the surgeon had made his investigation the patient was transferred to the medical service, for streptomycin therapy. Such a statement, of course, is made either to lead the discusser off the track or to direct him to a diagnosis of tuberculosis, because I cannot see that the patient would be sent to the medical service for streptomycin therapy unless there was reason to believe that he did in fact have tuberculosis.

I am not sure whether I am being led astray or whether this is a helping hand. I cannot believe that the lesion in the abdomen is tuberculosis. If it is, then I would assume that the patient has had a pancreatitis—which was the preoperative diagnosis; that he had latent tuberculosis, and that at the time the tuberculosis spread to the lungs in a miliary fashion. This means that he then had two diseases: (1) pancreatitis, and (2) miliary tuberculosis developing in the postoperative period. Against this is the fact that one of the first complaints was a cough. Hence it seems likely that despite the absence of clinical findings in the lungs, he had involvement of the lungs at the beginning of his present illness. So I am rather inclined to discount tuberculosis, unless perhaps as a secondary diagnosis, with pancreatitis as the primary one.

Now we come to the question of other types of malignancy. There are two possibilities:

6. LYMPHOSARCOMA
7. CARCINOMATOSIS

This may be a retroperitoneal lymphosarcoma, beginning in the upper abdomen and by virtue

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of pressure causing œdema of the gallbladder and the pancreas and finally leading to the adenopathy and splenomegaly.

It is well known that lymphosarcoma will involve the thyroid gland. Nodules of a lymphosarcoma have been found in the thyroid. This diagnosis would explain why there were glands behind the thyroid, but not why the surgeon removed the gallbladder. If this patient was suffering from retroperitoneal lymphosarcoma, it seems unlikely that the surgeon would have carried out such an operation.

The type of spread throughout the lung is not too common in lymphosarcoma. Nevertheless, one has to consider this as a probable diagnosis.

Then we come finally to the question of disseminated carcinoma. My inclination is to accept the diagnosis of carcinomatosis, despite the fact that we are dealing with a man 32 years of age. It is well known that when carcinoma does occur in younger people, the spread is rapid and many organs are involved. The question arises where it might be primary. One might presume that this man had a tumour of the head of the pancreas, of the ampulla, or somewhere in the biliary tract. This diagnosis would account for the œdematous pancreas, the distended gallbladder and in many ways the whole clinical picture. On the other hand, I think one must remember that carcinoma in the head of the pancreas, the ampulla or the biliary passages, if it does not lead to early jaundice, eventually does so before the patient dies. I think one is on thin ice in making a diagnosis of carcinoma in one or the other of these sites without the history that jaundice was at one time or another a feature of the disease. So I am not sure that I am on safe ground in making this diagnosis. Considering other structures in the upper abdomen, we are told the kidneys were normal, and the urine was normal except for a few red cells on one occasion. Might this be in the upper gastrointestinal tract—the stomach or the second part of the duodenum? There is a tumour of the stomach, an infiltrative carcinoma, the Jarcho tumour which invariably presents not as the primary lesion but from metastases. Metastases in the lung are very often the primary complaint, and the lesion in the stomach is not found until afterwards.

I can only assume that the surgeon dealing with the case was thorough, and when he said there was no disease of the stomach, duodenum or small bowel, we must accept his word for it. He is backed up by the radiologist, who also said he could find no disease of the upper intestinal tract.

I am getting rapidly to a point where I must find a primary for carcinomatosis, and am in the dilemma of trying to decide whether the patient has a lymphosarcoma or carcinomatosis due to a primary lesion in the biliary passages or pancreas. My inclination, despite the absence of jaundice, is to consider this to be a primary carcinoma, either in the biliary tract or in the pancreas, and that the lesions in the lung are a lymphatic spread of carcinoma from one or other of these sites. I cannot entirely exclude a lymphosarcoma, nor can I entirely exclude the possibility that this may have been a pancreatitis, with tuberculosis as a concomitant disease.

8. POLYARTERITIS NODOSA

There is one other disease which I have not mentioned and which has just occurred to me, which will give rise to protean manifestations and would explain the gastrointestinal complaints, the pain in the thigh and the miliary lesions throughout the lung—polyarteritis nodosa. I believe, however, that the lung picture is a little too florid for that diagnosis. There seem to be too many nodules. There are two further reasons. One is that people who have polyarteritis invariably have a history of asthma, associated with eosinophilia. Rackeman reported quite a number of cases, and in about 90% he found marked eosinophilia. But in this case there is no history of a bronchial spasm, asthma or eosinophilia, beyond 5%, and the patient has no evidence of kidney disease. The second is

CLINICAL DIAGNOSIS

Surgery:	*Medicine:*
1. Miliary tuberculosis—unproven, or	1. Pulmonary tuberculosis—hæmatogenous spread with C.N.S. involvement
2. Carcinomatosis—thyroid or testis.	2. Abdominal neoplasm (?)—pancreas, biliary tract or kidney (?)
3. Pneumoconiosis.	

DR. MILLS' DIAGNOSIS

Carcinomatosis—with lymphatic spread in the lung and the primary in the biliary tract or pancreas.

PATHOLOGICAL DIAGNOSIS

1. Carcinoma of the pancreas.
2. Carcinoma of the thyroid.

that most cases of polyarteritis eventually show evidence of renal disease. The absence of these features is noticeable in the case record, and against such a diagnosis. However, I would put polyarteritis down with the other possibilities.

FINAL DIAGNOSIS.—I finally came down to the diagnosis of carcinomatosis with lymphatic spread in the lung and the primary in the biliary tract or the pancreas.

PATHOLOGICAL DISCUSSION

*Dr. William H. Mathews**

This young man of 32 years was found at post-mortem examination to have a widespread carcinomatosis with much of the neoplastic tissue present in pinhead-size nodules superficially resembling a miliary tuberculosis, but not extensively affecting the serous membranes of the body cavities and without effusions in these cavities. He was grossly emaciated.

At the time, in 1949, and again upon review of the sections we concluded that this is an example of the by no means rare occurrence of multiple malignancy.

The history has indicated the known presence of a left lobe thyroid nodule at the time of discharge from the Army in 1945. This was a circumscribed tumour, encapsulated and of 3.5 cm. diameter, associated with metastases in the left cervical and supraclavicular lymph nodes—in other words, a malignant adenoma or acinar type of low-grade carcinoma of the thyroid with regional lymphatic metastases.

The abdominal exploration in 1948 revealed a diffuse œdematous and indurative thickening of the pancreas without other visible evidence of carcinoma.

In 1949, at the time of death, there was a diffuse indurative coarsening of the pancreas from head to tail, together with an expanded mass in the tail of the gland 5.0 x 5.0 x 3.0 cm. in size. Neoplastic, enlarged lymph nodes encircled the pancreas. This is interpreted as a small, acinar cell type of pancreatic carcinoma, diffusely involving the gland and associated, as is so frequently the case, with a chronic fibrosing pancreatitis.

The lungs were massively affected, in fact showing what amounts to a carcinomatous consolidation resembling pneumonia together with peribronchial and perivascular lymphatic involvement and with gross affection of pulmonary veins. Metastatic nodules were also present in the liver, kidneys, adrenal glands, splenic capsule, gallbladder, stomach, intestines, prostate, seminal vesicles, testes, urinary bladder, pituitary gland, thyroid gland, the heart, the brain and extensively throughout the bone marrow examined in the femur, two ribs and the spine at the two levels, first sacral and twelfth thoracic.

It might well be debated that this carcinomatosis all stems from a single primary source, say the thyroid tumour. We believe, however, that on morphological grounds there is good reason to suggest that the bulk of the carcinomatosis is of different cell type and that this is most probably primary in the pancreas.

Dr. Todd.—Dr. Mathews, was any evidence of tuberculosis found?

Dr. Mathews.—No.

Dr. Todd.—Dr. Pretty, you operated on this patient in November 1948. Would you tell us of the operation you performed.

*Dr. H. Gurth Pretty.**—For two weeks the patient was conservatively treated for pancreatitis. He failed to respond. The abdomen was explored. Chronic pancreatitis or carcinoma of the pancreas was suspected. In order to drain the pancreatic and biliary systems a cholecystjejunostomy was performed—in case the common bile duct should become obstructed by chronic pancreatitis or tumour. In the process of anastomosis of gallbladder to small bowel, the gallbladder was noted to contain cholesterol.

Dr. Todd.—In the 15 years 1940 to 1955, 4,252 autopsies were carried out at the Montreal General Hospital. Of these, 1,134 revealed carcinoma, and 48 of these were multiple. The number of cases of primary carcinoma of the pancreas has been 70.

On November 5, 1955, Dr. Harold Elliott gave a paper on sciatica at the Queen Mary Veterans' Hospital, and reviewed 1,000 cases between 1940 and 1954 in the Montreal General Hospital and Queen Mary Veterans' Hospital. One of the points he made was that pain in the thigh should always be kept in mind as possibly coming from carcinomatosis somewhere in the body. The patient under review complained of pain in the thigh at an early stage. From the pathology, he may well have had involvement of the lower spine at that time.

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Clinical and Laboratory Notes

RECURRENT PRIMARY VARICOSE VEINS

J. A. ELLIOT, M.D., F.R.C.S.(Eng.),
Vancouver, B.C.

THE TREATMENT of varicose veins has a very bad reputation both in the lay and medical mind. Many of the results justify such an opinion. Patients have even asked if they would be able to walk after the operation. Indeed the author had a case sent to him, following operation for varicose veins, in which the arterial tree had erroneously been ligated and stripped. Needless to say, the patient did not walk again on that limb, an above-knee amputation being necessary. In this presentation it is intended to outline some of the reasons why the treatment of varicose veins so often fails.

Firstly, one must define a varicose vein as a dilated incompetent vein. The adjective tortuous has been purposely left out of the definition because many varicose veins are not tortuous. There are two types of varicose veins, primary and secondary. The primary varicose vein develops from a vein which has a hereditary weakness and/or a congenital absence or deficiency of valves. The varicosity develops as a result of increased strain of a physiological nature such as prolonged standing, lifting, coughing, straining or pregnancy. Such strains in normal veins would not cause varicosities. Secondary varicose veins develop as a result of increased pressure of a pathological nature, as in deep venous thrombosis, postphlebitic syndrome, congenital and acquired arterio-venous fistulæ, abdominal tumours, and ascites.

In order to diagnose primary varicose veins one must think of and rule out the causes of secondary varicose veins, by far the commonest cause of which is the postphlebitic state of the deep venous system. In this condition there may be an incompetency of all three venous systems in the lower limb, namely, the superficial, deep and communicating. Because of this the Trendelenburg test, the only test of the many described which is of real value, gives a doubly positive result. The percussion test is of value in following the course of a vein, particularly in an obese patient. The Perthes test is occasionally valuable when there is a doubly positive Trendelenburg test but the history and physical findings do not suggest an incompetent deep venous system; in other words, where one suspects a competent deep venous system with incompetent communicators.

Before outlining the reasons for failure of varicose vein therapy, one should review the different methods that have been popular and are still being used by some of the profession as a primary form of therapy. Sclerosant therapy was first introduced by Pravaz in 1851; he used ferric chloride solution in an attempt to cure an aneurysm, and from then on sclerosant therapy gradually increased in popularity as a form of therapy for varicose veins so that by the late 1920's and early 1930's it was generally accepted. However, the results were so disappointing because of recurrence, and so often complicated by deep venous thrombosis, that it also was gradually discarded. At the beginning of this century, Babcock devised an intraluminal stripper and Mayo devised an extraluminal stripper. Both these forms of therapy lost favour in the 1920's mainly because of the popularity of injection therapy which could be performed on ambulatory patients without hospitalization. Sclerosant therapy failed because the thrombosed vein recanalized or collaterals developed around the thrombosed area. About this time, high ligation became popular and a great issue was made of the necessity of dividing all the branches at the upper end of the long saphenous. Because this failed, surgeons began to perform high and multiple ligations. Because there had been temporary benefit from the injection therapy, it was decided to practise high ligation and retrograde injection of a sclerosing solution—often large quantities were injected through a ureteral catheter as it was slowly withdrawn from the vein. The incidence of deep venous thrombosis was high; some patients died from pulmonary embolism and the morbidity from postphlebitic conditions was worse than from the original disease. In more recent years, surgeons have returned to the high ligation and stripping operation, but in many cases this procedure cannot be carried out because of marked tortuosity of the main vein, because of previous injection therapy, or even because of a previous multiple ligation operation. These difficulties can be overcome by excision of tortuous portions of the vein or by stripping short lengths of the vein through multiple incisions. Often very tortuous branches of the saphenous veins can only be dealt with by multiple ligations and subsequent injection therapy. So we may sum up by saying that the present-day peripheral vascular surgeon employs practically all the older methods but only after he has removed the main offending vein.

It is a well-known fact that if a vessel—be it artery or vein—is ligated, collateral vessels soon develop around the obstructed area. Another well-known fact, which applies to thrombosed veins but not to arteries, is that recanalization often takes place, as can be so often demonstrated in the postphlebitic vein. If we accept these two facts, we can immediately understand

why injection therapy, high ligation, multiple ligation, and high ligation and retrograde injection all failed as a primary form of therapy in primary varicose veins.

The anatomy of the long saphenous vein varies considerably, but in the great majority of cases it follows a fairly definite pattern. Beginning in the dorsal vein of the foot, it passes up in front of the medial malleolus and then runs obliquely across the lower third or half of the tibia; from here it runs obliquely to a point behind the medial femoral condyle and then passes in a slightly curved manner to the fossa ovalis, where it joins the common femoral vein. At its upper end are three or four important branches—the superficial external pudendal, the superficial inferior epigastric, the superficial external iliac circumflex and, most important of all when it is present, the accessory long saphenous.

The author believes that there are many reasons why the treatment of varicose veins fails, but that probably the commonest reason is failure of the surgeon to differentiate between primary and secondary varicose veins. The second commonest reason is that the surgeon has a stereotyped operation which he performs on all varicose veins; in other words, he tries to make one operation meet all the many variations seen. One reason for failure of high ligation—the development of collaterals—has already been mentioned. On many occasions, when operating on recurrent varicose veins, one has found that the ligation was performed one inch or more below the sapheno-femoral junction and all the branches at the upper end of the saphenous vein were left intact; on other occasions, the accessory long saphenous has been ligated in mistake for the long saphenous. Therefore during dissection of the upper end of the long saphenous vein, one must see the sapheno-femoral junction, and must clamp, divide and ligate all the branches in this area, ligate the long saphenous flush with the femoral vein, and must particularly search for the accessory long saphenous, though admittedly this is not always present. The author believes that the surgeon should have a good idea whether or not this vein is present before operation; to avoid missing it, he makes a practice of examining his patients the night before operation and carefully marking all the larger veins with Brazilian oil or gentian violet.

Another common reason for failure is that the surgeon fails to recognize a varicose short saphenous vein. One is not likely to misdiagnose varicosities limited to the short saphenous vein but, when there are primary varicose veins of both the long and short saphenous veins, it is quite a common error to fail to recognize the condition in the short saphenous vein. When the Trendelenburg test is carried out with the tourniquet below the knee, the result will be a singly positive test, but when the tourniquet is above the knee there will be an immediate filling of the incompetent short saphenous vein, giving a so-called doubly positive test. In order to be sure that this doubly positive result is due to an incompetent short saphenous vein, the Trendelenburg test must be performed with the tourniquet above the knee and simultaneous thumb pressure over the upper end of the short saphenous vein; a singly positive test will then be obtained in primary varicose veins.

CONCLUSION

The author believes that if primary varicose veins are properly treated the results are good; it is most unfortunate that the profession has allowed varicose vein surgery to develop such a bad reputation. Some of the reasons for this reputation have been given and suggestions made to correct the situation. The treatment of secondary varicose veins is an entirely different problem, and until those who operate on varicose veins realize this fact, the results will continue to be bad.

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THE FINER POINTS OF PERINEAL REPAIR

CZESLAW A. F. UHMA, M.D., *Sydney, N.S.*

A NUMBER of different methods are employed in maternity hospitals for perineal care during the puerperium, and opinions differ about the method which gives the best results. From discussions heard on this subject and from the many remedies for perineal pain advertised by pharmaceutical firms one might feel justified in concluding that the healing of perineal birth injuries is still a problem often encountered. Although the inflammatory complications which threaten, by infection, to disturb healing can be easily checked by the use of antibiotics, complaints of discomfort and soreness of the perineum in the post-partum period are still very common. In my opinion the various methods advocated for healing without discomfort are misdirected and do not affect the essential cause. This is the point which I would like to emphasize strongly—that healing and comfort after perineal repair depend primarily on the method of suturing, and that later perineal care has only secondary significance. If the suturing is correctly done, comfort in the perineal region during the puerperium is assured and only the simplest principles of asepsis need be observed.

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I should like to describe a method of repairing perineal tears or episiotomies which I have been using for many years and which I have only slightly modified since coming to Canada. From my teaching experience in Poland and later in Great Britain I can strongly recommend this method of suturing, and all who have used it have found it very satisfactory.

Depending on the conduct of the third stage of labour, suturing may be done before or after delivery of the placenta. There is no doubt a great advantage in packing the vagina with a single rolled pad before suturing, but the possibility of hæmorrhage, which may be concealed by the packing, must always be kept in mind and the condition of the uterus should be watched. General and local anæsthesia are equally suitable.

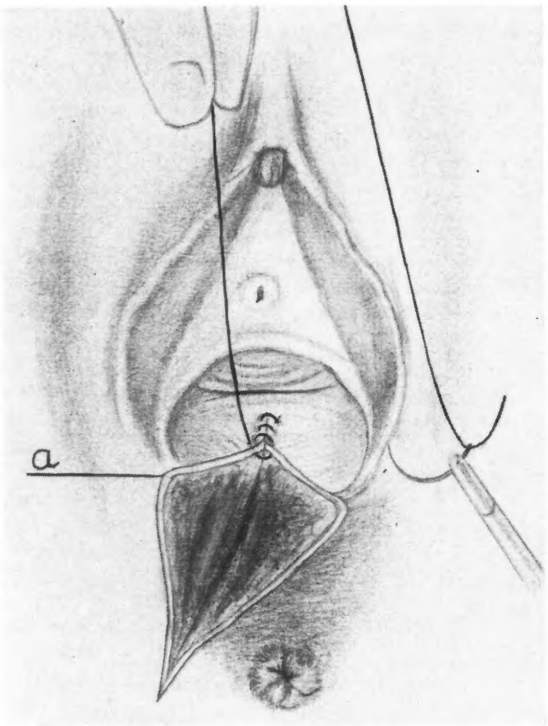

Fig. 1.—Approximation of vaginal wall. The continuous suture extends from apex of tear to commissure indicated by /a/ in diagram.

MATERIAL

Atraumatic 000 chromic catgut, or 00 plain catgut, is used. When ordinary sutures are used, a fine cutting needle is best for suturing of the skin.

PROCEDURE

Before beginning the repair it is advisable to exclude the anal area by a sterile drape.

First step: The two edges of the posterior commissure are seized with Allis's clamps. If this is not done, the surgeon should at least distinguish all landmarks along the edges of both sides which correspond, such as hymen, fourchette and posterior commissure of labium major, as well as corresponding folds, colour and pigmentation which would assist in making an accurate approximation. During suturing these landmarks should always be watched.

Second step: Starting from the upper end of the rupture or incision, a continuous suture of the posterior vaginal wall is carried out, taking about one-quarter inch (0.6 cm.) of the edge on each side, and leaving about one-quarter inch interval between stitches. The suture is continued downwards, watching on the way the landmarks which must be united, and is finished just at the commissure.

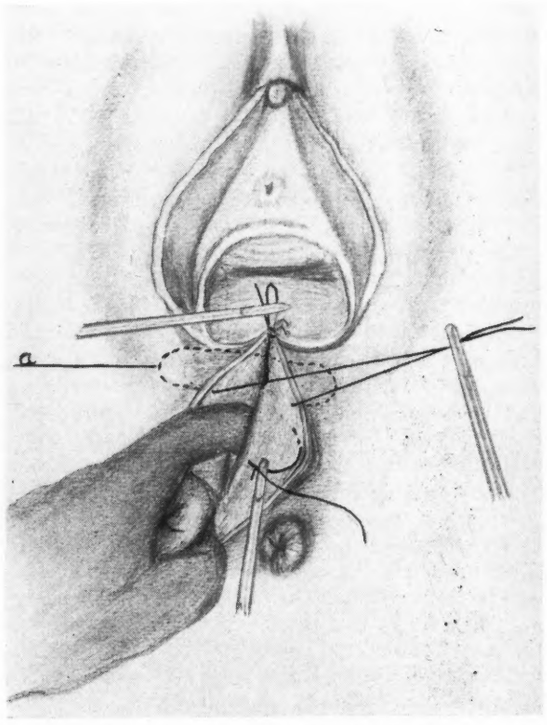

Fig. 2.—Sutures of muscles of perineal body. Suture at point /a/ is inserted quite deeply to pick up muscles retracted in direction of labium major of the side on which episiotomy is placed. The other suture is inserted under finger guidance, which also protects the rectum.

Third step: Insertion of two or three interrupted sutures in the levator ani and perineal muscles in a horizontal line. The first (upper) suture should be inserted more deeply in the direction of the labium major of the side on which the episiotomy is made, because of the marked tendency of muscles to retract forward along the labium. This is important in restoring the normal muscle balance of the perineum. The second and perhaps a third suture, if the episiotomy is long, should be inserted under control of the left index finger, which is placed between the separated muscles in the bottom of the

wound and palpates and directs the exact point of emergence of the needle and its insertion on the other side of the perineal body. The correct anatomical arrangement of the perineal muscles for suturing can be achieved when the free end of the continuous vaginal suture is pulled upward during insertion of these stitches.

Fourth Step: If the edges of the perineal skin are still far apart from each other, an additional continuous suture is started at the lower end of the wound. The purpose of this suture is to avoid dead space as well as to bring closer the skin edges. There is also another advantage of this

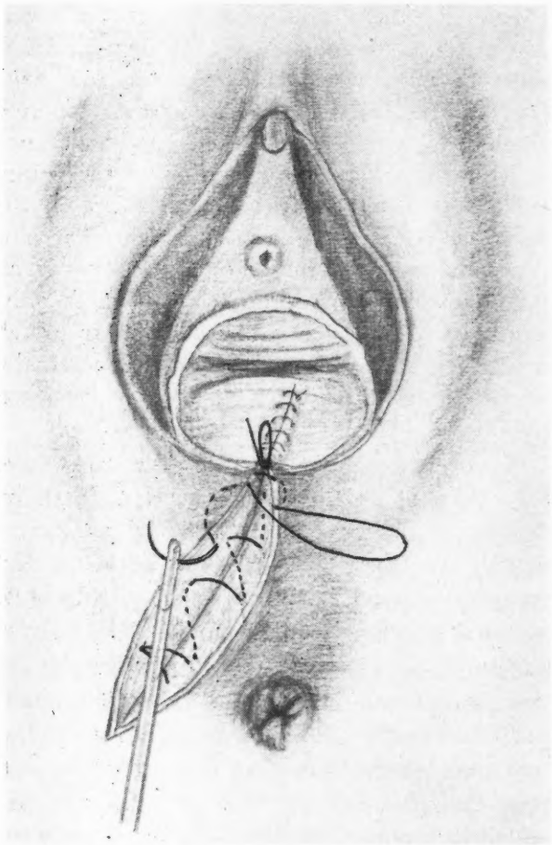

Fig. 3.—The additional continuous suture of perineal body has been inserted. After making a lock the same suture is used for final subcuticular closure.

stitch, namely that the thickness of the perineal body is built up. Without interruption the same stitch, after reaching the upper end, is continued as a

Fifth step: i.e., subcuticular suture of the perineal skin from upper to lower end of the incision. Alcohol swabbing finishes the perineal repair.

Throughout the repair the well-known precaution of avoiding undue tightness of the sutures should be observed.

The use of antibiotics such as Dicrysticin in one preventive dose seems to be advisable, especially in cases where any direct or indirect contact of the wound with the rectum cannot be excluded.

Each of the steps has special significance in prompt healing of perineal incisions, for example: (1) The continuous vaginal wall suture gives complete protection for the tissue of the perineal body while it is healing. The secretion of the vagina and lochia, which we know become full of pathogenic bacteria very quickly, cannot penetrate the vaginal wall sutured as above described, and the underlying tissue is completely protected from contamination. (2) In addition, this same suture, when pulled upward, brings the perineal muscles into exact anatomical relations in the perineal body and makes accurate insertion of the interrupted stitches under finger guidance easy. (3) A subcuticular suture gives the most desirable results in the perineal region—much better than in any other part of the body—and by its tightness gives further assurance that no infected material of vaginal or rectal origin will contaminate the healing tissues.

It has been my observation over many years that a perineum repaired in this manner is quite painless during the post-partum days, and as a matter of fact women usually do not realize that the perineum has been incised and sutured. When pain is complained of, one should suspect either that some infection is present or that some of the above details were not observed.

I wish to thank Dr. F. B. MacDonald for his suggestions and help in preparing this article.

BIZARRE CLINICAL MANIFESTATIONS OF HYPERTHYROIDISM

The diagnosis of hyperthyroidism is usually easily made. However, occult or "masked" forms of the disease are more common than is generally appreciated. Cases are described in detail where hyperthyroidism presented as abdominal pain, peripheral œdema, periarthritis of the shoulder, skeletal demineralization, severe myopathy, encephalopathy and epilepsy. Examples are also given of an excellent response of heart rate in congestive failure in the presence of hyperthyroidism, which is at variance with generally accepted medical teaching. Thyroid disease should always be suspected in cases of tendonitis or bursitis of the shoulder, as the association is very common.

Improvement in the above group of patients with proper recognition of the hyperthyroidism indicates that such a diagnosis should always be kept in mind, because incurable disease may be mimicked by a condition which is readily amenable to treatment.—E. M. Chapman and F. Maloof: *New England J. Med.*, 254: 1, 1956.

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AGAMMAGLOBULINÆMIA

During the past few years the procedures of electrophoresis and definitive fractionation of the serum proteins have led to the discovery and reporting of various syndromes resulting from the defective formation or actual absence of certain of these fractions within the human body. The major deficiencies of this type have included afibrinogenæmia or hypofibrinogenæmia, leading to coagulation defects; and agammaglobulinæmia, leading to various infective and immunological abnormalities. It is the latter condition with which we are particularly concerned here, not only because of its far-reaching implications with respect to infection, but also because of the knowledge it is likely to provide in connection with tissue-grafting.

The first reported case of agammaglobulinæmia was that of Bruton.[1] In 1952, he described the case of a boy who, between the ages of 4 and 8, had 18 bouts of infection with 8 different types of pneumococci, 5 episodes of otitis media and 3 attacks of mumps. After the administration of autogenous pneumococcal vaccine and typhoid vaccine, and after diphtheria immunization, no circulating antibodies could be detected. In this patient, a deficiency in serum globulin was demonstrated, and the recurrent infections were controlled by repeated injections of gamma globulin.

Since Bruton's original paper, and undoubtedly stimulated by it, several additional cases have been described[2-5] in the literature. While many of these cases differ in detail, a rather clearly defined pattern emerges on analysis. In almost all these patients there was a combination of: (a) a history of recurrent bacterial infections; (b) absence of acquired antibodies; (c) a lack of isohæmagglutinins; (d) an extremely low level (down to zero) of serum gamma globulin, although the total serum protein levels were within the normal range; (e) failure of long-term prophylactic antibiotic therapy to furnish protection; and (f) response to repeated protective injections of gamma globulin.

Though this syndrome has been well documented, there still remain certain differences of opinion among workers in this field. For example, there is some doubt in reported discussions whether associated conditions such as focal granulomatous processes in the spleen and liver[4] are the cause or the effect of the defective serum globulin formation. There is also some question whether all cases of agammaglobulinæmia are congenital, or whether the term may be applied to those cases in which similar effects are produced by general deficiencies in the serum proteins, *including* globulin.

However, it appears possible to resolve these differences of opinion, temporarily at least, by classifying cases of agammaglobulinæmia roughly into three categories. Those in the first group possess no antibodies and are subject to repeated infections with no apparent underlying disease. Those in the second group likewise appear to have no underlying systemic disease, but isohæmagglutinins are present, at least in low titre, and infections are not unusually frequent, even though other circulating antibodies are evidently produced in but very small quantity. In the third group are those who suffer from an associated systemic disease which might be regarded as either primary to or concomitant with the globulin defect. These patients possess isohæmagglutinins as well as certain fixed tissue antibodies, but are subject to repeated infections.

Cases of this serum protein defect are now being reported in Canada. At the 1955 Annual Meeting of the Royal College of Physicians and Surgeons of Canada, those present were treated to a very interesting discussion of this subject by Dr. W. A. Oille of Toronto, who described a case

of his own; and the ensuing discussion was most illuminating, ranging over not only the infective features of the syndrome, but also the immunological implications.

These immunological considerations are most interesting. It has been known for many years that a substantial obstacle to current medical and surgical progress is the established fact that tissues and organs from one person, transplanted to another, will not survive. The notable exception to this is the transplantation of corneal tissue; but it appears that corneal transplants represent a special case dependent on the isolation of the donor tissue from the blood and lymph of the recipient. Although it has been suspected that rejection of homografts has an immunological basis, proof of this concept has been lacking until recently, when Good and Varco[6] reported a successful homotransplantation of skin from a patient with agammaglobulinæmia to a burned child. At the time of submission of their paper for publication, the graft had been in place for 18 weeks and had taken well. This, of course, provides evidence in favour of the immunological theory of the rejection of homografts, and it also bids fair to be the first step in the clarification of many of the mysteries associated with repeated homotransplantation failures in the past. This is of special interest in connection with renal homotransplantations, because the kidney is probably the organ in which homografting has been most frequently attempted.

In any case, the current interest in defective serum globulin formation and the knowledge obtained from the study of this defect indicates that a great deal of further investigation is desirable. Furthermore, it seems clear that any patient who suffers from recurrent severe infections with any of the associated features already described should certainly have serum gamma globulin estimations carried out. It is very likely that we shall hear more about this most interesting subject during the next few years.

S.J.S.

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Editorial Comments

AURICULAR FIBRILLATION

The causes of auricular fibrillation are many, the most common being rheumatic heart disease and degenerative heart disease due to coronary atheroma and hypertension. Less common causes are thyrotoxicosis and constrictive pericarditis. Rare causes include pulmonary embolism, respiratory infections, administration of digitalis and electroshock therapy. In experimental auricular fibrillation, two mechanisms are possible.[1-3] (1) One focus may form impulses, but, owing to the rapid rate, the muscle cannot respond regularly. As a result these impulses spread irregularly over the atria and some of them are blocked—the "circus theory". More recently it has been shown (2) that stimuli can be rapidly formed in more than one focus, e.g. in the sinus and AV node. It is probable that the commonest form of clinical auricular fibrillation is the multicentric one, since rapid impulse formation in one centre induces rapid firing of impulses in others. In general, auricular fibrillation does not, if present without complications, endanger life. Even if it lasts for decades, no untoward effect will be noted, provided the ventricular rate is kept slow. The ultimate prognosis will depend upon the nature of the underlying heart disease. Symptoms and signs include dyspnœa, palpitation, anxiety, pulmonary œdema, pulmonary or systemic embolization, and loss of consciousness. However, there are certain advantages to well-established auricular fibrillation which include freedom from paroxysmal attacks with their attendant undesirable symptoms, the ease of control of the heart rate with digitalis and the comparative infrequency of the development of subacute bacterial endocarditis in these patients.

Fraser and Turner[4] discuss the pathogenesis of auricular fibrillation in rheumatic heart disease. They found that 30% of 500 patients admitted to hospital with auricular fibrillation had rheumatic heart disease. In a study of 500 patients with mitral valve disease, 43% had auricular fibrillation. They found no correlation between auricular fibrillation and the degree of mitral stenosis or the degree of pulmonary hypertension. However, there was a relationship between the degree of cardiac enlargement and auricular fibrillation. The question arises, does auricular fibrillation produce cardiac enlargement or do both exist because of a common cause? These authors were unable to correlate heart size with age, number of attacks of rheumatic fever, number of years since the first attack, or the degree of mitral stenosis. Cardiac enlargement presumably is due to a direct effect of the rheumatic process on the myocardium. Biopsy of the auricular appendage in patients undergoing mitral valvulotomy revealed Aschoff nodules in a higher percentage of cases with

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auricular fibrillation than in those with sinus rhythm. Mitral valvulotomy is not contraindicated in cases with auricular fibrillation; in fact it may improve the ultimate prognosis, but the risk is greater than in those with sinus rhythm because of increased myocardial damage, increased incidence of mural thrombi and hence increased risk of embolization. In the surgical group of 250 cases, intracardiac thrombosis was found in 40% of 106 cases with auricular fibrillation and in only 2% of 144 with sinus rhythm. Auricular fibrillation developed postoperatively in 23% of cases which had previously had a sinus rhythm. Quinidine preoperatively or postoperatively was of no value in preventing auricular fibrillation or treating it after it had occurred. Two weeks after operation, fibrillation was readily converted to sinus rhythm with quinidine in all but 3 of 60 cases. In general, quinidine is superior to procaine amide in the control of atrial arrhythmias.

N. W. McQUAY

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MENTAL RETARDATION

An interesting article appeared in a recent issue of the *Manitoba Medical Review* which really deserves some publicity outside the province. The Association for Retarded Children in Manitoba has been attempting to conduct a survey of mentally deficient and mentally retarded persons in Manitoba, of whom there are probably 12,000. It is a sad reflection on the present state of affairs that the whereabouts of many of these persons are simply not registered in some central place, so that appropriate organizations may render help. The Association is making contact with the medical profession in the hope of enlisting their co-operation in this search. This, in itself, is a forward step, for there is evidence that lay associations for retarded children have in the past been suspicious of professional persons, feeling that the latter do not have the real interests of these children at heart. The present association is first attempting to define areas in which the retarded child is being neglected, such as the education of pre-primary class children, or the provision of home visitors in caring for defective children. The next step has been the setting up of an experimental project in training, as a demonstration of what can be done.

The *Manitoba Medical Review* also published a brief and valuable survey of points on mental retardation which the general practitioner should know about. It is stressed that 3% of the general population are mentally defective. Of these, three-quarters are "educable", 20% are "trainable" and 5% are severely retarded. The importance is emphasized of not making a definite diagnosis to the parents until it is as certain as possible, even if this means waiting until the child is of school age. In this way, months or years of hardship for the parents may be avoided. As reading material for parents the article recommends "The Backward Child" published by the Federal Department of Health and Welfare. For the physician, the WHO Technical Report No. 75 "The Mentally Subnormal Child" is recommended. Other points made are that the defective child, particularly under school age, needs the security, affection and acceptance of a good home unless he is to become emotionally as well as intellectually handicapped, and that the physician should acquaint himself with the legal provisions for the care and protection of mentally defective persons in the province. The article ends by quoting another authority as follows: "The doctor's chief aim is to encourage the parents to adapt themselves to the child's handicap, and the child to make the most of his resources."

MALNUTRITION IN ACUTE AND CHRONIC ILLNESS

During the past decade, the concept of masked undernutrition has received a great deal of attention from practising physicians and research workers alike. Most physicians have had the unfortunate experience of seeing certain of their patients fail to recover from their illnesses and gradually "fade away", although their treatment has followed all the recognized precepts and has appeared in all other respects to be satisfactory. The suggestion that such treatment-failures were the result of masked undernutrition became popular during the Second World War, when carefully controlled studies were carried out in Canadian military hospitals to determine what proportion of the food presented to patients on hospital trays was actually ingested. It came as no surprise to many students of this problem that a large proportion of the hospital diet presented to patients was not being accepted; and that, despite the routine provision of adequate amounts of food, this food was not being eaten.

Almost concurrently, the importance of adequate protein nutrition in acute and chronic illness was being stressed by students of metabolism in various Canadian universities. It soon became quite clear that, in any acute insult to the body economy, whether infective or traumatic, a state of negative nitrogen balance rapidly ensued, which was most difficult to correct and which required a degree of protein intake amounting almost to saturation, if it were to be corrected at all. It was suggested that the uncorrected state of negative nitrogen balance was responsible for a high proportion of treatment failures, particularly in traumatic and non-traumatic surgical cases; and a great deal of additional study was given to this problem. An endocrine basis for this situation was intensively sought and soon found; and it finally became accepted that the adrenal cortex, responding in its customary manner to acute injury, was the culprit by virtue of its well-known protein catabolic effect. It was also suggested that other hormones such as testosterone, which are known to have a distinct protein anabolic effect, might be used in an attempt to correct this undesirable state of affairs.

The matter appears to have been left at this stage for the past few years; and, although physicians have been using this knowledge to a certain degree, the original flurry of interest has subsided and many of the basic principles have been forgotten. In the original work on this subject, alluded to above, most of the problems were solved satisfactorily as they applied to acute illness; but no such satisfactory explanations were available to explain the malnutrition that occurs in certain non-malignant chronic illnesses which do not specifically affect the gastrointestinal tract. Goodman and Dowell,[1] in a very thoughtful study, have recently added a great deal to our information on this very perplexing problem. In their paper, 138 cases of undernutrition in 432 chronically ill patients are reviewed in detail. These writers go to great lengths in describing the physical findings in their patients suffering from undernutrition. These included hepatomegaly, dental defects, pallor, weakness, decubitus ulcers, muscle atrophy, mucous membrane abnormalities, abnormal mental states, œdema and diarrhœa. Surprisingly enough, with the exception of a mild normocytic normochromic anæmia, the basic laboratory data in these undernourished individuals were within normal limits. It is possible that more complex studies such as measurements of 17-ketosteroid excretion and serum vitamin levels might have revealed abnormalities; but these were not done, and have no particular bearing on the problems that are being posed by these workers.

In general, and this is stressed by the writers, there are two methods of correcting undernutrition of this order of severity. These are, firstly, the use of adjuvants, such as vitamins, testosterone, corticosteroids, or appetite stimulants, which are mentioned only because they were found to be of little value; and secondly, the provision of adequate nutrition *combined with a serious attempt to ensure that this nutrition is actually accepted.* This latter feature requires close and continued supervision on the part of physicians and nurses if it is to meet with any degree of success. It should be noted that some workers in this field have had a modicum of success by tube-feeding, particularly in patients whose disease or injuries precluded feeding by any other means. However, not only was this not the problem in this particular study, but it was unsuccessful in a large proportion of patients when instituted. The writers indicate that "many refused to swallow the tube, others removed it repeatedly, and, in general, for one reason or another, the tube feeding method gave unexpectedly poor results. The same poor results were obtained in gastrostomy cases." It appears, therefore, that there is no simple mechanical method of ensuring adequate nutritional intake in anorexic patients; and that close attention to food intake by attending hospital staff, both physicians and nurses, is mandatory.

Perhaps the most striking feature of the statistics collected in this study is that, excluding psychiatric causes and unexplained anorexia, the leading basic cause of malnutrition proved to be poor eating habits of long standing; and it is possible that, on closer study, the majority of patients with unexplained anorexia would have fallen into the "poor food and eating habits" group. In view of the fact that outstanding and often dramatic results in respect to weight gain and improvement in general well-being were obtained simply by adequate attention on the part of the hospital staff to the food intake, it would certainly appear that poor eating habits were the major cause of the undernutrition noted. Furthermore, although the writers stress the fact that nutritional problems of adults are often traceable to poor childhood eating habits, they emphasize that this is not a sufficient reason to gloss over the nutritional problems of the adult.

The entire subject leads one to ponder on the rather strange situation into which we have been led by the food habits inherent in our present western civilization. In adult life, a large proportion of our population finds it necessary, for reasons of health, to undergo weight reduction because of excessive food intake in childhood—while another important segment of the population suffers from clinically evident undernutrition, equally traceable to poor childhood eating habits. S.J.S.

REFERENCE

1. GOODMAN, J. I. AND DOWDELL, W.: *Ann. Int. Med.*, 43: 1241, 1955.

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PUBLIC RELATIONS FORUM

Conducted by L. W. HOLMES,
Assistant Secretary, C.M.A.

XII. THE DOCTOR SPEAKS

ONCE THE BLUEPRINT of the medical society's PR programme has been drawn, the next step is preparation of the specifications—the details of the plan. This is essential to successful implementation of the programme.

There are several activities which a society may consider under the suggested blueprint heading, *Information and Education.* Among them are:

1. Improved press relations.
   (a) Medical-press code of co-operation.
   (b) Medical-press conferences.
   (c) Establishment of good media contacts.
   (d) Adequate pressroom facilities at annual meetings.
2. Radio and/or television programmes.
3. Active Speakers' Bureau.
4. Health Forum.

These activities help to meet many medical PR needs. They display to the press a sincere desire to eliminate misunderstanding, to help the press in its news-gathering tasks; they provide a medium for health education and the telling of medicine's story; they help prove that doctors are not shirking their responsibilities in the field of health education. And finally, they introduce the doctor to many laymen who have little or nothing to do with the profession.

The foregoing places considerable emphasis on health education. But leadership in this area falls into the domain of the medical profession, and it is a responsibility which doctors must willingly accept. There is a strong and growing public demand for information on medical subjects. Whether or not the doctor views this as undesirable, newspapers, radio, TV and magazines all recognize the demand and attempt to meet it. Unfortunately much of the material these agencies produce is classified as health "miseducation". But it need not be; the profession can guide the media of public information in the presentation of medical news, and it can also take this information direct to the public.

There are at least four effective and controllable means by which to reach the public with health information: the speakers' bureau, the health forum, the radio and television programme, and the profession-sponsored newspaper health column.

The following is a discussion of the first of these—the speakers' bureau.

The speakers' bureau is an organized medical society plan to provide local lay groups with physician-speakers who will speak on medical and scientific subjects.

Having reached the decision to establish a speakers' bureau, the medical society's next and probably most difficult task is to obtain participants. There will be many refusals on the grounds of lack of free time or inability to speak in public. The generation of enthusiasm for this PR project requires a concerted selling campaign among society members, stressing the value of the speakers' bureau to self, colleagues, profession and community.

There are two ways in which potential speakers may be canvassed. A list of proposed topics can be sent to members with the request that they place their names alongside those subjects on which they feel qualified to speak. Or the members may be asked to suggest topics on which they would be willing to speak. In either case, the doctor should also be asked to indicate when he could most conveniently accept speaking assignments—morning, afternoon or evening —and the most suitable days of the week.

Of course, not all doctors are silver-tongued orators, but ability to speak in public can be developed. Some medical societies in the United States have organized accelerated public-speaking courses. They draw on speech instructors from local high schools, vocational schools or universities to participate in such courses.

It is essential that the doctor speak in a language which his audience will understand. Since most of the groups to which he speaks will be composed of laymen, he must reduce technical terms to lay equivalents, or be prepared to explain any technical language he may use.

Who wants to hear the physician speaker?

Almost every organization in the community represents a potential audience: service clubs, school and church organizations, study groups, labour organizations, women's clubs.

When the speakers' bureau has been organized, subjects selected and assigned, and speakers trained, the next step is to secure the audience. This can be accomplished by sending out letters and leaflets to the groups in the community outlining the service, listing topics and giving details of how speakers may be obtained. This might be supplemented by newspaper stories.

It must be remembered that many clubs make up their programme for the entire season in advance, usually in the late spring or early summer. The society's bid for participation in those programmes, therefore, must be placed with the group in time to be considered by the programme committee.

The society should also point out that the service is free and that alternate topics should be requested in anticipation of a speaker's being unable to participate when scheduled.

Additional public relations value can be obtained from the speakers' bureau if the public is kept informed of its activities. When a speaker has been requested to address a local group, the newspaper should be informed giving the speak-

er's name, his topic, his audience, and the date, time and place of the appearance. This provides an advance notice of the meeting, helping to assure a good audience, and at the same time publicizes the efforts of the local society. Newspaper coverage of the talk may also be stimulated in this manner.

The speakers' bureau is a precursor of other health education projects—the health forum, and radio and television programmes; these will be discussed in subsequent articles.

AMERICANS LOOK AT THEIR DOCTOR

Last year the American Medical Association sponsored a nationwide survey of the public's opinion of doctors. The results were obtained from personal interviews with 3,000 members of the public, 500 physicians in private practice, and 100 persons in each of five special groups—editors, columnists and commentators; lawyers; pharmacists; registered nurses; and executive secretaries of state and county medical societies.

The following covers the major findings of the survey as reported this month in an A.M.A. news release:

1. Most Americans have their own family doctor—82%. Ninety per cent of rural farm dwellers have their own physicians, and high percentages were also found among white-collar workers, middle-income, college-trained people, and central state residents.

2. Most people like their family doctor, and like doctors as a group. Ninety-six per cent gave an unequivocal "yes" reply when asked, "Do you like your own doctor as a person?" They cite as the reasons: personal interest, sympathy, kindness, competence, intelligence, a friendly personality and manner, frankness and honesty. They gave the same reasons, with some exceptions, for liking doctors generally. However, given an opportunity to criticize through true-or-false questions, the difference in attitude in evaluating doctors generally and family physician appeared:

Doctors don't give patients as much time as patients would like—about "other" doctors, 60% say "true"; about "my" doctor, 18% true.

Most doctors try to hide other doctors' mistakes—54% true (not asked about personal physicians).

Doctors are hard to reach for emergency calls—other doctors, 51% true; my doctor, 19%.

Doctors are not frank enough—other doctors, 46% true; my doctor, 15%.

They charge too much—other doctors, 43% true; my doctor, 16%.

They think they are always right—other doctors, 43% true; my doctor, 23%.

They keep people waiting too long—other doctors, 41% true; my doctor, 15%.

They don't have enough personal interest in patients—other doctors, 39% true; my doctor, 11%.

Their charges have gone up faster than other living costs—other doctors, 35% true; my doctor, 13%. These figures were about the same for the statement that doctors make too much money compared to their patients.

3. The public has a realistic idea of the number of years of training required to practise medicine, and the length of their own doctor's work week. With respect to training, the answers ranged from seven to 11 years or more. The public's median estimate of their doctor's work week is 64 hours, with 12½% of this time given to charity work. The report said that the doctors' own estimates are 62½ hours and 13% charity.

4. Doctors are more critical of themselves than are other people. Their agreement that some of the listed complaints are true ranges as much as 20% above the public's.

They closely agree with the public on estimates of their capability, intelligence and willingness to accept medical advances, and feel most appreciated for competence, sincerity, and healing abilities. They are quicker than their patients to agree that they do not give as much time as they would like, and on lack of availability for emergencies.

5. When people criticize physicians, it is largely for the cost of care; they do not, however, think doctors are trying to "get rich quick".

6. They are evenly split for and against "sliding scales" of fees.

The A.M.A. reports that, with few exceptions, the findings about the total public apply also to sub-groups: men and women, young and old, white and blue collar, and geographically different groups. Some minor differences, tending to be less favourable, are seen in people over 65, non-whites, Southerners, low-income groups, non-high-school-educated persons, and those without personal physicians.

The percentage differences in opinion between union and non-union persons are almost indistinguishable, the report concludes.

GENERAL PRACTICE

CERTAIN ASPECTS OF RENAL DISEASE IN CHILDHOOD*

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DIAGNOSIS MUST ALWAYS precede therapy. Before discussing renal diseases in children, one should carefully consider certain points in the interpretation of the urinalysis since this forms the basis of diagnosis. It is surprising how many children have had an incorrect diagnosis because of lack of understanding of the significance of the urinalysis.

URINALYSIS

Taking the urinary specific gravity is still the best clinical method for determining renal function, provided one recognizes certain limiting factors. One must be certain that the urinometer is properly calibrated. This can be tested by placing it in distilled water at room temperature. It should read 1.000. The maximum specific gravity of urine varies with age. For the first six months of life a normal infant may not be able to concentrate above 1.018. After this, most children can concentrate urine as well as the

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adult. With one exception, if these limiting factors are used, a urine of high specific gravity implies good renal function. This one exception is the presence of a non-reabsorbable solute in the urine. For clinical purposes only glucose and albumin will appear in the urine in sufficient amounts to alter the specific gravity. This will occur only with mellituria and the nephrotic syndrome. If one eliminates proteinuria and glycosuria, high specific gravity of the urine indicates normal renal function.

A low urinary specific gravity means either that there is failure of tubular reabsorption of water or that the patient is adequately hydrated. The tubular failure may be organic or functional. The usual organic causes are pyelonephritis or chronic nephritis. Functional failure of water reabsorption may be from lack of the pituitary antidiuretic hormone, or from a functional failure of the renal tubules to respond to ADH. This can be differentiated by the Hickey-Hare test.[1] A concentration test in an œdematous patient must be interpreted cautiously since delivery of œdema will invalidate the test.

Proteinuria, except in orthostatic proteinuria, always signifies renal disease. The renal disease may be functional, as in patients with a low renal plasma flow. This is the usual cause of proteinuria in the dehydrated or febrile patient. If the proteinuria disappears promptly with rehydration, this is the likely etiology. It is rarely feasible to get figures for 12-hour protein excretion in children. One usually has to rely on qualitative tests. This merely measures the amount of protein in that specimen. A small amount of protein in a small volume of urine may give a markedly positive test, whereas the same amount in a large volume may be negative. An estimate of the urinary volume can be made if the specific gravity is known. A low specific gravity suggests a large volume; a high specific gravity, a low volume. If the specific gravity is low and protein is present, one should suspect organic renal disease. If the specific gravity is high, the proteinuria may represent functional changes. The classic error is seen in the patient with chronic renal disease. As the disease progresses the specific gravity decreases and with this the qualitative proteinuria decreases. Frequently this is interpreted as improvement in the renal status, whereas it may be evidence of impending renal failure.

Errors in interpretation of the microscopic examination of the urine are equally common. An Addis count would be desirable but is rarely practical. To adequately appraise a centrifuged urine one must know the volume of urine, and the time and speed of centrifugation. A large volume with a long and fast centrifugation will suggest an abnormal sediment just as the converse may indicate a normal sediment. Few physicians will have equipment for accurate centrifugation, so that I feel it is wise to learn the evaluation of uncentrifuged specimens. In my experience this has not led to erroneous diagnoses. The urine should be examined shortly after voiding. In hospital practice this is the commonest source of error. With a low specific gravity or an alkaline urine, red cells hæmolyze and casts break up rapidly. Again the specific gravity plays an important role. Pathological numbers of formed elements may be excreted, but with a low specific gravity may be broken up or diluted sufficiently to be missed. A high specific gravity usually means oliguria. The normal patient excretes considerable numbers of red cells, white cells, and casts. If the urinary volume is low, one may find these elements and think the patient has renal disease. In the presence of a high specific gravity, particularly if the patient is febrile or dehydrated, the discovery of a slight increase in the formed elements may be of no significance. Disappearance of these elements on rehydration of the patient should suggest that the patient does not have organic renal disease.

Elevation of the blood urea nitrogen (BUN) or non-protein nitrogen (NPN) means diminution of glomerular filtration without differentiating between organic or functional origin. Nitrogen retention associated with a low specific gravity suggests organic involvement of the glomeruli. Nitrogen retention and a high specific gravity suggests diminution of renal plasma flow rather than organic renal disease.

ORTHOSTATIC PROTEINURIA

The frequency with which children with orthostatic proteinuria are labelled as chronic nephritics and assigned to the role of an invalid is distressing. There is widespread agreement that orthostatic proteinuria is seen frequently in adults. Little has been written on the subject as it occurs in children. Thus few physicians think of it when they find proteinuria in a child. In the past 8 years we have studied 18 children, all of whom had been diagnosed as chronic nephritics, with marked orthostatic proteinuria. In the follow-up to date, none has developed evidence of renal disease in spite of full activity. The following tests gave normal results: concentration test, urinary sediment, hæmogram, sedimentation rate, BUN, urine cultures, and intravenous urography. A typical patient was a girl who had been at bed rest for eight years because the proteinuria cleared when she was at bed rest. Instead of recognizing the diagnosis the physician interpreted this as a favourable effect on chronic renal disease. Eight years later she is well and the proteinuria has ceased.

An adequate work-up can be completed in 24 hours. A voided specimen is obtained at the time of admission. The child is promptly put on absolute bed rest. After two to three hours the patient, if female, is catheterized. Cultures and urinalysis are done on this specimen. In a male a clean catch specimen is obtained within five

minutes of assuming the erect position. Fluids are restricted until morning. The bladder is emptied at 10.00 p.m. to eliminate bladder urine which might contain protein. The first voided specimen in the morning before arising is tested for the specific gravity and protein. BUN level and sedimentation rate are determined and the patient is sent for intravenous urography while still dehydrated. All information can be obtained within this period. If the orthostatic test is unequivocal and the remainder of the studies are normal, urography probably is not necessary.

PYELONEPHRITIS

Symptoms of pyelonephritis in infancy rarely involve urinary complaints. The commonest symptoms are fever, vomiting, and diarrhœa. Any combination of these symptoms or recurrent fever alone should suggest the possibility of renal infection. Dribbling or a weak urinary stream suggests the possibility of obstruction. In my experience dysuria is usually associated with vaginitis rather than pyuria. Rarely is enuresis a symptom of pyelonephritis. Gross anomalies of the abdominal musculature are frequently associated with renal anomalies.

The diagnosis of pyelonephritis in the male is relatively simple. A clean catch specimen with more than six white cells per low-power field suggests the possibility of infection, and a clean catch specimen is then cultured. In the female a voided specimen is of value only if negative. Nearly all voided urines in females contain white cells derived from the external genitalia. If white cell casts are found, this is proof of the renal origin of the infection. If white cells alone are found in the female, catheterization must be done if one is to avoid making an erroneous diagnosis. All bladder urines with abnormal numbers of white cells should be cultured in order to differentiate infection from the increase in white cells seen in dehydrated children.

Once the diagnosis of infection of the urinary tract has been made, the biggest problem is when to ask for urological help. Certain findings warrant urographic study with the *first* clinical attack. Dribbling, an abdominal mass, a gross defect of the abdominal musculature, and persistence of proteinuria or hypertension are signs which demand a urographic study. Any patient having a second proven attack should have a similar study.

Recently we have seen four children with recurrent abdominal pain and unexplained fever who have had persistently normal urinalyses. They were found to have hydronephrosis with complete obstruction of the pelvic outlet. Probably children with this picture should have pyelography.

Treatment of the patient with obstruction is obvious. In recent years we have been seeing a group of children with normal renal systems who have had recurrent pyelonephritis. There is no clear answer to the management of these children, except the prolonged use of appropriate antibiotics for a period of years.

ACUTE GLOMERULONEPHRITIS

There is general agreement that approximately 95% of children with acute nephritis will recover without therapy. For some reason a 5% mortality rate in a medical disease does not seem to worry many doctors. A surgical mortality rate of this degree would be considered most serious. Since there is excellent evidence that the mortality in acute nephritis can be reduced almost to zero, every case of acute nephritis should be considered an emergency. Almost all deaths in acute nephritis occur during the first 14 days of the illness and are associated with hypertension and its complications, cardiac failure and encephalopathy. If the hypertension is controlled, cardiac failure rarely occurs. In almost all of the patients hypertension can be controlled with either magnesium sulphate or one or a combination of the newer antihypertensive agents. When used properly, magnesium sulphate will lower the blood pressure in a majority of the patients. Most pædiatricians believe that magnesium sulphate is ineffective when given orally. Intramuscular injections are effective, but painful and potentially toxic. I feel that it should be used as one uses digitalis, i.e. to effect. A 50% solution given intramuscularly in dosages of 0.2 c.c./kg. is effective. If this fails to lower the blood pressure in two hours, it is repeated as frequently as every three hours if the diastolic blood pressure stays above the desired levels. In children over six years of age we like to keep the diastolic pressure below 95 mm. Hg. In younger children we like to keep the diastolic pressure below 90. We have not recognized magnesium intoxication, but are always ready to give calcium gluconate if evidence of intoxication should appear. We use intravenous magnesium sulphate only when the patient is having continuous convulsions. A 1% solution is given slowly and stopped as soon as the convulsion has ceased.

Recently Daeschner has reported that reserpine is effective when given intramuscularly in single dosages of 0.08 to 0.15 mg./kg. Etteldorf has found that the combination of Apresoline and reserpine is effective. He used Apresoline in dosages of 0.1 to 0.15 mg./kg and reserpine, 0.07 mg./kg. Their reports suggest that either schedule is as effective as magnesium sulphate. They are less painful and have little toxicity.

The importance of controlling the hypertension explains why most children with acute nephritis should be observed in the hospital. Any child with nephritis who develops dyspnœa or a headache should be immediately admitted to hospital.

There is no good evidence which would suggest that diet, prolonged bed rest, or prolonged use of prophylactic antibiotics has any effect on

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the ultimate course of the disease, which nearly always goes on to complete healing once the hypertensive course is over. In general we keep the patient in the hospital until the blood pressure has been normal for three days without therapy. The patient is then kept at bed rest at home until the urine is grossly clear. This usually occurs within six weeks after the onset of the disease. The patient is kept on limited activity at home until the urine and sedimentation rate are normal, at which time he is released to full activity. Several patients have gone back to school while still showing evidences of activity of the disease. These have done as well as those patients on a more restricted regimen.

REFERENCE

1. LITTLE, J. M., YOUNT, E. H. AND KELSEY, W. M.: *Am. J. Med.*, 14: 41, 1953.

Association Notes

DOMINION INCOME TAX RETURNS BY MEMBERS OF THE MEDICAL PROFESSION

[*We publish herewith the text of a memorandum approved by the Department of National Revenue for the guidance of doctors making income tax returns relative to the year 1955.*]

As a matter of guidance to the medical profession and to bring about a greater uniformity in the data to be furnished to the Taxation Division of the Department of National Revenue in the annual Income Tax Returns to be filed, the following matters are set out:

Individuals whose income—(a) is derived from carrying on a business or profession (other than farming); (b) is derived from investments; or (c) is more than 25% derived from sources other than salary or wages, are required to pay their estimated tax by quarterly instalments during such year. Each payment must be sent in with Income Tax Instalment Remittance Form T7C Individuals. Any balance of Income Tax due is payable with interest on or before the 30th April of the succeeding year.

Doctors who pay salaries or wages to employees are required to deduct tax therefrom in accordance with the Table of Tax Deductions obtainable from District Taxation Offices. Each employee should complete and file one copy of Form TD1 with his employer (a) at commencement of employment and (b) within seven days of any change in circumstances affecting his personal exemptions. If Form TD1 is not filed, tax deductions must be made as though the employee were a single person. Tax deductions withheld from salaries or wages must be sent to the local District Taxation Office not later than the 15th day of the following month accompanied by Tax Deduction Remittance Form TD7A.

The following timetable indicates the returns required:

A. Doctors NOT receiving salaries amounting to ¾ of income:

Date due	*Forms to be used*
March 31	Form T7C Individuals
April 30	Form T1 General
June 30	Form T7C Individuals
September 30	Form T7C Individuals
December 31	Form T7C Individuals

B. Doctors receiving salaries amounting to ¾ or more of income:

Date due	*Forms to be used*
April 30	Form T1 General (Note: Doctors whose earned income consists solely of salary and whose investment income is not over $2,400 may use Form T1 Short unless they claim a capital cost allowance or a foreign tax credit.)

C. Doctors who pay salaries to their own employees:

Date due	*Forms to be used*
15th of each month	Form TD7A
February 29	Form T4 Summary and Supplementary

Details of the total salaries or wages paid to employees and the tax deducted therefrom must be forwarded to the local District Taxation Office on Form T4 Summary and T4 Supplementary not later than the last day of February in each year.

INCOME

Under the provisions of the Income Tax Act a doctor is required to maintain an accurate record of all income received both as fees from his profession and by way of investment income. The record should be clear and capable of being readily checked against the return filed. It may be maintained on cards or in books kept for the purpose. Such records must not be destroyed until written permission for their disposal is obtained from the Minister of National Revenue.

EXPENSES

Under the heading of expenses, the following accounts should be maintained and records supported by vouchers kept available for checking purposes:

(a) Medical, surgical and like supplies.

(b) Salaries or wages paid to professional assistants, nurse, office help, bookkeeper. (It is to be noted that the Income Tax Act does not allow as a deduction a salary paid by a husband to a wife or vice versa. Such amount, if paid, is to be added back to the income.)

(c) Telephone expenses (long-distance charges on business calls and service charges for business telephones listed in the doctor's name, fees for telephone answering services).

(d) Assistants' fees; the names and addresses of the assistants to whom fees are paid should be furnished. This information is to be given each year on Income Tax form known as Form T4, obtainable from your District Income Tax Office.

(e) Rentals paid. The name and address of the owner (preferably) or agent of the rented premises should be furnished [see (i)].

(f) Postage and stationery.

(g) Depreciation or capital cost allowance as it is referred to in the Income Tax Act; a description of the treatment of depreciation may be found on page 4 of the Income Tax Return Form T1 General under Part XI Method.

The method of computing depreciation for tax purposes is the same as that used last year and you should have no difficulty if you have a copy of last year's return available.

Simply carry forward the balance remaining in each class after deducting last year's allowance. Add to this figure the cost of any new equipment purchased and deduct the proceeds from any disposal of property in

each class. The rate you wish to use not exceeding the maximum rate (see below) is applied to this new balance for each class to obtain the depreciation you may claim this year.

The maximum rates for the classes of equipment used by doctors follow:

Capital Item	*Class*	*Annual Maximum Depreciation*
Medical equipment		
(a) Instruments costing over $50 each and medical apparatus of every type	8	20%
(b) Instruments under $50 each	12	100%
Office furniture and equipment	8	20%
Motor car	10	30%
Buildings of frame construction	6	10%
Buildings of brick construction	3	5%

Where a doctor practises from a house which he owns and resides in, the allowance may be claimed as above on a portion of the cost of the residence, excluding land. For example, if the residence were a brick building costing $12,000 and one-third of the space were used for the office, the doctor would use $4,000 as the business portion of the cost and apply the building rate of 5% to determine the maximum depreciation allowable in the first year.

For further information on the subject you may refer to the Income Tax Regulations or you may consult your District Taxation Office.

(h) Automobile expense (one car). This account will include cost of license, oil, gasoline, grease, insurance, garage charges and repairs.

The capital cost allowance is restricted to the car used in professional practice and does not apply to cars for personal use.

Only that portion of the total automobile expense incurred in earning the income from the practice may be claimed as an expense and therefore the total expense must be reduced by the portion applicable to your personal use.

(i) Proportional expenses of doctors practising from their residence

[a] owned by the doctor: where a doctor practises from a house which he owns and as well resides in, a proportionate allowance of house expenses will be given for the study, laboratory, office and waiting room space, on the basis that this space bears to the total space of the residence. The charges cover taxes, light, heat, insurance, repairs, capital cost allowance, and interest on mortgage (name and address of mortgagee to be stated).

[b] rented by the doctor: only the rent and other expenses borne by the doctor such as heat and light will be apportioned inasmuch as the owner takes care of other expenses.

The doctor should be prepared to demonstrate, if called upon to do so, that his apportionment of any particular item is in accordance with the facts relative to that item.

(j) Sundry expenses. These should cover only small items not otherwise classified, for example, laundry, malpractice, insurance, etc. The expenses charged to this account should be capable of analysis and supported by records.

Claims for charitable donations should be made in the space provided for this item on the Income Tax forms and should not be included in the professional expenses. Such claims are allowable as a deduction from income up to 10% of the net income upon submission of receipts to your District Taxation Office.

The annual dues paid to governing bodies under which authority to practice is issued and membership association fees, to be recorded on the return, will be admitted as a charge. Initiation fees and the cost of attending postgraduate courses will not be allowed.

(k) Carrying charges. The charges for interest paid on money borrowed against securities pledged as collateral security may only be charged against the income from investments and not against professional income.

(l) Business tax will be allowed as an expense, but Dominion, Provincial or Municipal income tax will not be allowed.

Convention Expenses

Contrary to its practice for previous taxation years the Taxation Division has advised that claims for convention expenses cannot be admitted for 1955 and subsequent taxation years under the law as it now reads. This ruling follows the decisions handed down by the Income Tax Appeal Board and the Exchequer Court in the appeals of Dr. H. Griffith. In both cases it was held that convention expenses were not a proper deduction from income for income tax purposes. The Taxation Division must apply the findings of the Court. We are continuing in our efforts to convince the Honourable the Minister of Finance of the necessity for recommending an amendment to the law which will permit the deduction of these expenses.

Professional Men under Salary Contract

The Income Tax Act provides that income from an office or employment is liable to tax without deduction of any kind except such as are specifically provided for in the Act. The allowable deductions include the employee's contributions to a pension fund, alimony, travelling expenses, annual professional membership dues, office rent, salary to an assistant or substitute, and supplies consumed directly in the performance of the duties of employment.

Section 11 (10) (a) of the Income Tax Act permits the deduction from income of an office or employment of annual professional membership dues only if their payment "was necessary to maintain a professional status recognized by statute" and if their payment was "required by (the) contract of employment".

The annual registration fee of the Provincial medical licensing authority would be allowable if paid by the doctor himself.

Certain conditions are attached to the allowance of the expenses and without trying to recite the exact provisions of the law the main points are:

(a) That the expenses must have been incurred in the performance of the duties of the office or employment.

(b) That the employee is required, under the contract of employment, to pay the expenses.

(c) To claim travelling expenses the employee must be ordinarily required to carry on the duties of his employment away from his employer's place of business. Travelling between the doctor's home and his office is not included.

Where travelling expenses are allowable under these provisions, depreciation may be claimed on the automobile used for this purpose but no other claim for depreciation may be made.

Income from a Partnership

Additional expenses incurred by a partner, but not charged to the partnership, may be claimed as a deduction from the partner's share of income. However, the partner must be in a position to substantiate these expenses, to show why they were not charged directly to the partnership and that they were necessarily laid out to earn the partnership income.

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LAVAL UNIVERSITY AND QUEBEC MEDICINE

WHEN THE ANNUAL MEETING of the Canadian Medical Association takes place in Quebec next June, a great contribution towards the success of the congress will undoubtedly have been made by those connected with the Faculty of Medicine of Laval University. For over 100 years this university has been responsible for medical education in the city of Quebec, and it is of some interest to trace the history of medical education in the ancient city before and during the life of the university. It would seem that the first person to practise medicine in the neighbourhood of Quebec City was Jacques Cartier's barber-surgeon, whose chief claim to fame lies in the excellent autopsy account he gave of a case of scurvy. Many years later Champlain no doubt brought medical assistants with him, and we hear that one of them, Bonnerme, was involved in a plot against the leader's life. In the early days of the French régime surgery was the first of the arts and crafts to be subjected to some organization, and the first surgeon in the new colony was a Dieppe man, Adrien Duchesne, who apparently stayed on in Quebec during the brief occupation by the English under Kirke. During the 17th century the only means of medical education was by apprenticeship which was regulated from 1660 on. Apprentices served for three to five years and the usual pattern was a family one, the art passing from father to son. In this way whole dynasties such as the Soupiran family arose and continued, sometimes for over a century. The first Canadian-born surgeon was Louis Maheut, born in 1650, who after his apprenticeship studied in Paris and returned to Canada. It would seem that our medical predecessors in Quebec had a thin time of it financially. Fees were low and we have on record that in 1729 a governmental financial inducement was forthcoming to persuade a surgeon to go and live in the impoverished town of Trois-Rivières. Presumably the history of state medicine in Canada dates from that moment. Surgeons were not uncommon during the French régime, and charlatans also abounded, but the number of physicians was strictly limited. Between 1608 and 1763 there were only three of these rather superior and considerably less practical gentlemen in Quebec City. During the 18th century the lieutenant of the King's first

Service de Ciné-Photographie, Office Provincial de Publicité, Québec.

The older portion of Laval University. The Medical School was opened in September 1854.

surgeon was charged with the first examination system for licensing of surgeons, and we hear of a vain attempt by one Jourdain Lajus to create a closed shop in Quebec City and limit the number of surgeons to four. After the entry of the British into Quebec various attempts were made to regulate the practice of medicine. The law of 1788 gave state control of licensing, but certain unsatisfactory features led to its amendment in 1831. The system was still one of apprenticeship for five years with examination by an elected board.

Early attempts in the 19th century were made to create a medical school in Quebec City. The first in 1819 failed; the second at the Marine Hospital in the thirties was more successful, though we hear that the teaching of human anatomy, as in other cities, was only sustained by the efforts of the resurrection men. By the time the medical school of Quebc was established in 1848, the city had already got its first medical journal and its first medical society. Stress was at this time laid on the need for a knowledge of both official languages, and it is recorded that two candidates failed their final examinations because they were not bilingual. The medical school of Quebec several years after its formation made a relatively painless transition into the Medical Faculty of Laval University, which was established in 1852 by the grant of a Royal Charter to the seminary originally opened in 1663. The Medical School itself opened in September 1854, at least sixty years after the first plan for a university had been suggested and turned down. The inaugural lecture in 1854 was given by a remarkably progressive physician, Jean Blanchet, the first dean, who had studied in London and in Paris and whose inaugural address might in many respects serve today, with its emphasis on the need for a close acquaintance with the basic sciences. From the start Laval aimed at really high standards. Persons with a knowledge of the classics were favoured and the entry requirements were the stiffest on the continent of America. Laval graduated its first doctor, Larue, in June 1859; on this day this extremely well-educated man, whose great interest was forensic medicine and toxicology, read his thesis on suicide. In its early days Laval was singularly fortunate in its medical deans, and it is worth noting that the third and fourth of this line, Drs. James Sewell and Alfred Jackson, were both Protestant loyalists with an Edinburgh background. In 1876 after complicated negotiations a branch of the Laval Faculty of Medicine was created at Montreal, but this alliance seems to have led to considerable difficulties and in 1883 both the parent and the branch were menaced with closure, mainly on economic grounds. Assistance from Rome prevented such an unfortunate event. In 1889 the Montreal branch became independent of Quebec, except for the granting of degrees, and in 1919 the last ties were severed.

Men were versatile in the old days of Laval. Dean Lemieux, who directed the Faculty from 1885 to 1899 and was a generous and kindly man, had held the chairs of anatomy, physiology, pathology and legal medicine. Among the faculty giants of the first half century we might mention Michael-Joseph Ahern, the brilliant surgeon of the Hôtel-Dieu, who was one of the first to take up antisepsis, and Dr. Charles Verge, of an old loyalist family, whose charity was such that he used to shepherd an old drunk to confession every Saturday and eventually ended by curing him.

The seventh dean, Laurent Catellier (1905-1910), was a surgeon of some wit. One of his *bons mots* was "Make haste to use this treatment while it's still effective." He saw the University through a worrying time when the subject of a central examining authority in medicine was being hotly discussed; before his tenure of office ended, the medical course had gone from four to five years.

The history is one of progress and development of the curriculum, with the greatest upsurge in research under the tenth dean, Arthur Rousseau, a stern disciplinarian, who held office from 1921 to 1934, remodelled and stiffened the curriculum, ushered in the affiliation with schools of nursing and strengthened the ties with France. From then on, there was a steady increase in hospital beds and laboratory facilities. There are now six general and six special hospitals associated with the Faculty, and the latter itself envisages a transfer to a much larger new building on the plateau of Sainte-Foy, near the Ecole de Commerce where the June scientific programme will take place.

When you visit the C.M.A. Convention in June, you will have a chance of making closer contact with this historic medical school. In our next issue, we will say something about the hospitals of Quebec.

MEETING OF THE TCMP COMMISSION

Under the chairmanship of Dr. H. H. Lees of Windsor, the semi-annual meeting of the Trans-Canada Medical Plans Commission was held in the Executive headquarters, Toronto, January 27 and 28, with representatives of all Member organizations present.

Among other reports the Commission heard recommendations from the Committee of Plan Medical Directors on the development of Service

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Classification Manuals for Medical Services, and from the Committee of Plan Administrators on matters related to extensions of coverage and other activities and proposals associated with national correlation and extension of the prepayment programmes.

Considerable attention was given over to the question of research activities and proposals considered for the establishment of a joint Research Bureau in co-operation with the Canadian Medical Association, which would be devoted to developing statistical and other research for the joint needs of the Member Plans and the medical profession. A committee under Dr. M. R. MacCharles of Winnipeg was appointed to investigate further and report on this matter at the June meeting.

Among other activities a special committee was appointed under Dr. S. A. Orchard of Saskatoon to consider the further development of a National Uniform Contract to the level of comprehensive coverage.

Following other general business, arrangements for the Annual Meeting at Quebec in June were left in the hands of the Executive Committee.

ARE YOU GOING OVERSEAS THIS YEAR?

CANADIAN DOCTORS proposing to visit the United Kingdom can greatly assist the B.M.A.-C.M.A. currency exchange programme by depositing dollars with the C.M.A. and collecting pounds from the B.M.A. on arrival.

This plan has operated for several years with the approval of the Bank of England and for the fiscal year 1956 it will permit two British doctors to visit this country. The rules are simple; two Canadian doctors may send their cheque to the Canadian Medical Association in the amount of $560 and the B.M.A. will provide £200 when they reach the United Kingdom. In turn, we will furnish dollars to the two medical visitors identified to us by the British Medical Association when they arrive in Canada.

This arrangement is much appreciated by the visiting British doctors who would otherwise have difficulty in financing their travels. We are grateful to those C.M.A. members who have participated in the past and we invite anyone who desires to take part this year to communicate with the General Secretary at 150 St. George Street, Toronto 5.

MEDICAL SOCIETIES

THE MONTREAL PHYSIOLOGICAL SOCIETY

The third meeting of the year was held on Monday evening, January 16, in the Medical Building, McGill University. Attendance may not have been up to our usual standard (Mr. Rudolf Bing of the Metropolitan Opera, New York, was speaking on the same campus), but this was certainly not evident in the discussions following each presentation—they were lively and intense.

The Mechanism of the Goitrogenic Effect of Sodium Chloride; by Henri Isher, Department of Anatomy, McGill University.

The mechanism by which sodium chloride enhances the goitre produced by a low iodine diet in mice was investigated. Evidence was brought forward that sodium chloride did not cause goitre by enhancing the secretion of thyrotrophic hormone or the response of the thyroid gland to that hormone. Nor did it interfere with the iodine metabolism within the thyroid gland, as antithyroid drugs do. The data supported the hypothesis that sodium chloride primarily increases the iodine losses into the urine, thus decreasing the amount of iodine available to the thyroid gland. The resulting decrease of thyroid hormone secreted would account for the stimulation and eventually the greater size of the thyroid gland.

The Effect of Glucagon on Metabolism of Glycine-1-C^{14}; by N. Kalant, Jewish General Hospital.

Fasted intact rats were injected with glycine-1-C^{14} and glucose-1-C^{14}, and the effects of glucagon and epinephrine on incorporation of the C^{14} into liver glycogen and expired carbon dioxide were studied.

(a) The rate of incorporation of C^{14} from glycine into liver glycogen was increased by a previous injection of glucagon, and decreased by a previous injection of epinephrine.

(b) The rate of incorporation of C^{14} into glycogen was decreased, while the incorporation into CO_2 was increased by concurrent injections of glucagon.

(c) The incorporation of C^{14} from glucose into liver glycogen was increased by a previous injection of glucagon.

(d) Glucagon appears to cause a "rebound" accumulation of liver glycogen comparable to that produced by epinephrine.

Extraction and Assay of Tyrosinase from Frog Skin; Influence of Melanophore Hormone on Synthesis of the Enzyme;* by John L. Purvis and Orville F. Denstedt, Department of Biochemistry, McGill University.

Tyrosinase from the skin of the frog (*Rana pipiens*) was made amenable to extraction in good yield by exposing skin slices to 0.5% trypsin. The partially digested subcutaneous tissue was removed with a scraper and the skin was homogenized in water, centrifuged at 3,500 r.p.m. for 20 minutes, and again at 18,000 r.p.m. for 20 minutes. The supernatant fluid contained the enzyme. The method developed for assaying the enzyme involved measurement of oxygen consumption in the Warburg apparatus with an excess of crystalline tyrosine in the medium to ensure saturation. Oxygen consumption and melanin formation, as measured by the optical

*This study is supported by a contribution from Canada Packers Ltd., Toronto.

density at 540 mμ, were found to be proportional to the amount (dry weight) of the enzyme preparation. That the reaction is enzyme catalyzed was established by the following criteria: (a) heat inactivation; (b) indispensability of Cu ++ ions; (c) inactivation on addition of copper-binding agents. Studies on the specificity of the enzyme indicated that it is primarily a monophenolase.

The study has revealed a pronounced seasonal variation in the enzyme activity of the frog skin, the activity increasing about tenfold during the fall months. Serial injections of the melanophore hormone or a subjection to dark adaptation for a period of two months failed to increase the activity of the enzyme. A. H. NEUFELD

CANADIAN ASSOCIATION OF RADIOLOGISTS

The Canadian Association of Radiologists held its 19th Annual Meeting in Vancouver on January 16-18, 1956. Dr. Andrew Turnbull of Vancouver succeeded Dr. Ronald Burr of Kingston as President. Dr. Guillaume Gill was elected Honorary Treasurer, replacing Dr. D. L. McRae, of Montreal.

The meeting was well attended and very successful, being highlighted by Dr. William Boyd's presentation of the Richards Memorial Lecture. His subject was "Spontaneous Regression of Cancer". Dr. J. H. Woodruff of Los Angeles, California, and Dr. C. T. Dotter of Portland, Oregon, were visiting speakers. Panel discussions on both diagnosis and therapy by representatives of many branches of medicine were well received.

At the business meeting there was much discussion on national health insurance. The federal government and most of the provincial governments have announced that diagnostic services will be made available to the public. The Canadian Association of Radiologists concluded that the present high quality of diagnostic services can be maintained by assisting already established services in universities, hospitals and doctors' offices. The Association believes that separate government diagnostic centres would be costly and unnecessary. The Association nominated two members to the Canadian Medical Association Liaison Committee with the Department of National Health and Welfare.

The Association adopted the International Recommendations on Radiation Protection as the Canadian Code and suggested that they be summarized for publication in the *Canadian Medical Association Journal*. Items of particular importance refer to the hazards of fluoroscopy by unqualified persons, the dangers of shoe-fitting fluoroscopes and the hazards of handling radium, radon and radioactive isotopes.

ALCOHOLISM RESEARCH MEETING

The Alcoholism Research Foundation of Ontario held a most stimulating and informative meeting of research workers on January 27 and 28 at their premises in Toronto. The object of the meeting was not so much the presentation of formal papers on various aspects of alcoholism as the exchange of information on an informal basis and the discussion of projects already in progress or contemplated. Since much of the work described was not yet complete, it would be premature to give a detailed account. We hope, however, to have the opportunity of publishing reports of projects of particular interest to clinicians at a later date in the Journal. In addition to the staff of the Alcoholism Research Foundation, there were visitors from Ontario, Quebec and the United States (Dr. Ebbe Hoff, Medical Director, Division of Alcohol Studies and Rehabilitation, Virginia, Dr. Robert Straus, Syracuse, New York, and Dr. J. M. Ruegsegger). The first day began with discussions of subjects in the fields of pharmacology, physiology and biochemistry, including investigations of new drugs. The meeting then continued with presentations in the area of sociology, psychiatry and psychology.

From the volume of research reported, it would seem that much dead wood has been cut away and that enough promising work is in progress to ensure a continued good contribution from Canada towards the solution of problems associated with alcoholism.

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

A NEW POLIOMYELITIS VACCINE

With a flourish of press trumpets and a minimum of technical information, the new Minister of Health has announced that a new Salk-type of poliomyelitis vaccine has been evolved in this country. The only official information that has been divulged as to its constitution is that it is prepared from three strains of virus, the type I, or Mahoney, strain having been replaced by one of less virulence. There are grounds for believing that this less virulent strain is related to the Brunhilde strain. It was also announced at the Minister's press conference that introduced the new vaccine that it is planned eventually to replace types II and III with milder strains. The vaccine has been produced by two British pharmaceutical companies working in co-operation with the Medical Research Council. According to *The Lancet*, it is understood that the vaccine produced by one of these companies will contain attenuated strains of types II and III as used by Sabin.

The new vaccine will not be available in substantial quantity until the end of the year, but limited supplies will be available during May and June, which it is hoped will be sufficient to inoculate 250,000 to 500,000 of the 5,500,000 children aged 2 to 5 years in England and Wales. For the present the vaccine will only be available through local health authorities, and no inoculations will be allowed during the poliomyelitis season—July to October inclusive. The government proposes at the outset to buy all the vaccine that can be produced. At a later stage, after November, it is hoped that it will be possible to extend the age-groups to be offered vaccination, and general practitioners will then be given an opportunity to participate in the scheme.

VISITING CHILDREN IN HOSPITAL

A survey in 1954 showed that the number of hospitals which found it possible to allow daily visits to children had trebled during the previous two years. This meant that only 10% of hospitals admitting children still restricted visiting to one day a week or less, including 28 hospitals, compared with 142 in 1952, which prohibited all such visits except in emergencies. The Minister of Health has now issued a memorandum to all hospital boards and committees urging them to reexamine the

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position. Not only is the hope expressed that daily visiting will become the general practice, but the suggestion is made that visits might even be allowed more frequently than once a day. Special attention is devoted to the problem of daily visiting in infectious diseases hospitals, and the essential safeguards are outlined. Total prohibition of visiting is recommended in the case of certain diseases—e.g., smallpox, typhoid fever, and dysentery—and children of school age should be allowed to visit only in exceptional circumstances.

The Vagaries of National Prescribing

An analysis of the National Health Service prescribing figures in England for 1954, recently published in the *Pharmaceutical Journal,* draws attention to the marked variations which occur in different parts of the country. The figures for 1954 were: average total cost per prescription 50.36*d.* (range, 41.31*d.* to 60.5*d.*); average frequency per person 5.41 (range, 3.45 to 8.86); average total cost per person 272.6*d.* (range, 154*d.* to 418.75*d.*). In the industrial north the total cost per person was very high and the frequency per person was high, but the total cost per prescription was low. In the seaside resorts cost per prescription, frequency and cost per person were all high, whereas in the dormitory counties around London the average cost per prescription was high, the frequency was moderate and the total cost per person was low. The conclusion is reached that high frequency of prescription rather than high cost per prescription is responsible for the high cost per person. In other words, patients get large numbers of prescriptions of cheap medicines in some areas, and a smaller number of expensive medicines in other areas. "And it seems to cost more to give a lot of cheap medicine than a smaller quantity of expensive medicines." The final conclusion is one which will not appeal to the Ministry of Health in its persistent campaign to discourage the prescribing of proprietary medicines: "It would probably be found that the use of branded medicines is not associated with unduly high cost per head. In other words, that the intelligent use of proprietary medicines can be a means of reducing the drug bill."

Therapeutic Deaths

The Registrar General's detailed analysis of the vital statistics of England and Wales for 1952, which has just been published, gives details of the 149 deaths due to medical or surgical treatment which occurred in that year. There were 84 males and 65 females, and 121 of the deaths occurred in general hospitals, 15 in mental hospitals, none in nursing homes and 13 elsewhere. Air embolism was responsible for 12 deaths. Of the 24 deaths due to adverse reactions to drugs, four were related to penicillin and one to streptomycin. Other drugs involved were chloramphenicol, mesantoin, methylthiouracil and thiosemicarbazone—all causing agranulocytosis. Of 14 deaths of psychotic or psychoneurotic patients, two were associated with operations on the brain, five with insulin treatment and seven with electroconvulsive therapy. During the three-year period, 1950-52, there were 1,874 deaths attributed to anæsthetics. Thiopentone was responsible for 208 (11.5%) of the 1,799 deaths in which the name of the anæsthetic was given. In these cases thiopentone was the only anæsthetic used. In a further 458 cases (25.5%) thiopentone was one of the anæsthetics administered. Of local analgesics, "nupercaine" was associated with 20 deaths. Between them, procaine, "novocaine", "novutox", "planocaine" and "duracaine" were associated with 29 deaths.

WILLIAM A. R. THOMSON

London, February 1956.

ABSTRACTS *from current literature*

MEDICINE

Hepatitis in Mononucleosis.

R. J. HOAGLAND AND R. T. MCCLUSKEY: *Ann. Int. Med.,* 43: 1019, 1955.

It has been known for some time that liver involvement is not a complication but a regularly occurring feature of infectious mononucleosis. This is not surprising, since the cause of infectious mononucleosis appears to stimulate proliferation of lymphocytes, and the mesenchymal cells of the liver are potential precursors of lymphocytes. The realization that acute hepatitis is regularly present in infectious mononucleosis has been followed by the unwarranted assumption that this acute viral hepatitis should be managed like other viral diseases of the liver. However, it does not appear logical to recommend identical therapeutic regimens for two viral diseases merely because they affect the same organ.

The primary purpose of this investigation was to ascertain whether histological studies would or would not support the belief, based on clinical experience, that the hepatitis of mononucleosis is mild, and that therefore early ambulation is unlikely to be harmful. To this end, needle biopsies of the liver were performed in a succession of seven unselected patients with infectious mononucleosis to investigate the degree of hepatic damage. Liver biopsies were also performed on three infectious mononucleosis patients with jaundice to compare the hepatic histopathology of such patients with that of non-jaundiced patients. These findings were compared with the hepatic pathology of infectious hepatitis. All of the biopsies were performed with the Vim Silverman needle and stained in the usual way with hæmatoxylin and eosin. All of the liver biopsies in cases of infectious mononucleosis showed abnormalities in the form of lymphocytic infiltration, with only minimal hepatocellular changes and without alteration of hepatic architecture. The pathology in the patients with and without jaundice was essentially the same. In other words, this investigation showed that the hepatitis of mononucleosis is distinctly different from infectious hepatitis, and is characterized by lymphocytic infiltration, minimal hepatic cell damage and no architectural change.

Accordingly, there is histopathological support for the *liberal* management of mononucleosis patients with and without jaundice.

S. J. SHANE

The Course of Idiopathic Pleural Effusion in Fifty Patients.

J. T. SKAGGS AND G. W. SMILEY: *Am. Rev. Tuberc.,* 72: 647, 1955.

Fifty patients with so-called "idiopathic pleural effusion" were studied; the clinical features are outlined. In 29 cases aspirated fluid was examined for tubercle bacilli by culture, guinea pig inoculation, or both. Only seven diagnoses of tuberculosis were made by these methods. It is believed that tubercle bacill would have been found more often *if more cultures of larger quantities of fluid had been made.*

Follow-up studies were completed in 42 of the 50. The cumulative incidence of tuberculosis was 16% and five patients died, thus making a case mortality rate of 10%. The fact that each of these patients consulted his physician because of specific complaints shows again the important role of the family physician in tuberculosis case finding.

The writers think that idiopathic pleural effusion is almost invariably a manifestation of tuberculosis and should be so treated.

S. J. SHANE

The Relationship of Cirrhosis of the Liver to Hypertension: A Study of 504 Cases of Cirrhosis of the Liver.

H. F. LOYKE: *Am. J. Med. Sc.*, 230: 627, 1955.

A number of investigators have commented on the absence of experimental and clinical hypertension with liver disease. Recent studies have tended to confirm these ideas, when it was found that the frequency and degree of hypertension were substantially higher in a control group than in patients with subacute liver atrophy who later came to post-mortem examination. It was concluded that hypertension could not occur in the presence of more than minimal liver damage. In view of this report, the author sought to examine the nature and frequency of hypertension in a consecutive series of cases with cirrhosis of the liver, obtained from the records of two large general hospitals; 504 cases with a positive diagnosis of hepatic cirrhosis were studied.

The study confirmed the impression that hypertension is less likely to occur in cirrhotics than in the normal. Nevertheless, the renal and neurogenic mechanisms capable of elevating the blood pressure still remain intact in the presence of hepatic cirrhosis. In some cases, hypertension can co-exist with at least moderate hepatic disease, but tends to disappear as hepatic failure advances. It was at first thought that hypertension was less common in the presence of cirrhosis because the damaged liver is unable to form the protein substrate on which renin acts. Certain findings in this study, however, cast doubt on this theory. However, a large proportion of cases in which the blood pressure fell as the liver failure advanced, constitute most impressive evidence of the dependence of "essential" hypertension on adequate hepatic function. It could be argued that these declines in blood pressure were the result of progressive malnutrition, cachexia or salt restriction. However, there is no evidence of this from the clinical records.

It is concluded, therefore, that hypertension does not usually occur in the presence of cirrhosis; that hypertension may exhibit itself in cirrhotics *if* renal disease develops; that stressful stimulation may produce temporary hypertension in cirrhotics; and that established hypertension may disappear with the onset of hepatic cirrhosis.

S. J. SHANE

"Open Healing" of Tuberculous Cavities. Incidence and Pathology in 240 Resected Specimens.

J. R. THOMPSON: *Am. Rev. Tuberc.*, 72: 601, 1955.

While it is commonly supposed that epithelization of the lining of cavities is in part a manifestation of "open healing", it has been pointed out that epithelization of an "open-healed" cavity is the exception rather than the rule. While some authorities argue that a cavity is not healed as long as it remains open, the disappearance of the tuberculous elements and replacement by fibrous tissue *does* constitute healing of the disease process *per se*. It has been suggested that the pathological criteria for "open healing" should consist of sharp demarcation of the cavity by fibrous tissue, absence of tubercle bacilli in the wall, and the disappearance of caseation or other tuberculous elements. Before the use of present-day antimicrobial agents, it was most unusual to see tuberculous cavities with evidence of "open healing". Because of the increasing incidence of this peculiar type of healing in surgically resected pulmonary tissue, all of the resected specimens from 335 consecutive cases of pulmonary tuberculosis in a municipal tuberculosis sanatorium obtained from 1948 to 1954 have been reviewed. The results of this analysis form the basis of the present report. In 23 cases, "open healing" of tuberculous cavities took place. This represents 9.6% of all the cavitary lesions. While this is a significant figure, one should not lose sight of the fact that the remaining 90.4% still showed the common variety of chronic tuberculous cavity. Of the 23 patients, 20 had been treated with isoniazid with dihydrostreptomycin and PAS or streptomycin and PAS, and the cavities could easily be differentiated from bronchiectatic or emphysematous lesions.

The fact that none of the specimens resected before 1952 showed evidence of "open healing" substantiates the belief that chemotherapy is a predominant factor. The pathogenesis of this form of "open healing" is discussed.

S. J. SHANE

SURGERY

Gallstone Ileus.

S. L. DECKOFF: *Ann. Surg.*, 142: 52, 1955.

The proportion of cases of mechanical intestinal obstruction caused by gallstones is about 1-2%, and it may be decreasing with the increasing proportion of postoperative adhesions and greater frequency of cholecystectomy for silent gallstones, but the average general surgeon will have several cases during his lifetime. It occurs once or twice a year in a large hospital or about once in 30,000 operations; it is a disease of advanced age and the mortality is high—up to 50%.

Stones might enter the intestinal tract by ulcerating through the gallbladder wall, into the peritoneal cavity, and thence ulcerating the intestinal wall, or via the ampulla of Vater, but most ulcerate from gallbladder to intestine by a fistula which might heal. Less than half the stones entering the gastrointestinal tract will obstruct it, for they must be quite large. Only about half the cases give a history of suggested cholelithiasis, for it is the large single stone that is often responsible for the obstruction. Acute cholecystitis preceding the intestinal obstruction occurs in about 25%, and jaundice in 10%. A history of recurrent partial obstruction is common before the complete obstruction. The signs vary, depending on the site at which the stone becomes wedged, but the commonest are those of the terminal ileum. Occasionally an intragastric stone may cause intermittent pyloric obstruction. A correct preoperative diagnosis of the cause of the intestinal obstruction is unusual, but it should be suspected in small bowel obstruction in an elderly woman who has had no previous laparotomy and no hernia. Fever and leukocytosis are not characteristic, but a careful radiological examination may be of great help. Many of these patients are diabetic. A long intestinal tube may give great preoperative relief, but tends to delay operation too frequently in these elderly obese patients.

Operation is the only proper treatment, and if possible the stone should be extracted by incising the bowel. The one death in the 12 cases reported was due to overhydration.

BURNS PLEWES

Injuries to the Trigeminal Nerve, its Ganglion and its Divisions.

G. JEFFERSON AND J. SCHORSTEIN: *Brit. J. Surg.*, 42: 561, 1955.

In reporting 66 new cases of injuries to the trigeminal nerve in its intracranial and bony course, an impression is given that such lesions are not rare and should be detected more often. Sixteen of the lesions were ganglionic, seven caused by gunshot wounds. The commonest damage was to the infraorbital nerve, comprising almost half the total. Usually it is caused by a fracture of the maxilla through the inferior orbital canal and is manifested by numbness of the upper lip. Injuries to the nasociliary nerves are infrequent.

Recovery of sensation in ganglion injuries is uncommon and painful paræsthesiæ may persist.

BURNS PLEWES

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The Resectability of Gastric Carcinoma.

I. BOEREMA: *Ann. Surg.*, 142: 228, 1955.

It is agreed that operations for carcinoma of the stomach should often be more radical, and that they should be operations for cure rather than for palliation. The radical gastrectomy advocated removes the whole stomach, part of the first portion of the duodenum and of the lower œsophagus, the tail of the pancreas and the gland-bearing omenta. A segment of jejunum joins the œsophagus to the duodenum. A plastic instrument has been devised, on the principle of the Murphy button, to accomplish an anastomosis from below the diaphragm without stenosis. It is removed from above. The stomach is removed as a closed bag.

This technique was used at the Wilhelmina Gasthuis in Amsterdam, and resulted in an 80% resectability rate in operations for gastric carcinoma. The operative mortality was 15% in patients ranging from 37 to 79 years of age. Compared with the partial gastrectomies done by the same surgeons previously, the resectability rate rose from 43% to 80% and the operative mortality did not rise.

BURNS PLEWES

Some Remarks on the Repair of Flexor Tendons in the Hand with particular reference to the Technique of Free Grafting.

A. B. WATSON: *Brit. J. Surg.*, 43: 35, 1955.

Correct procedure is important in treating hand injuries, and this is emphasized before actual technique is discussed. Tendon divisions should not be repaired during the first visit to the casualty department unless: (1) The wound is of such a type that it is sure to heal by first intention. (2) Main operating room facilities and good anæsthesia are used. (3) A surgeon dealing regularly with tendons does the operation personally. Otherwise, the wound alone should be repaired and the tendon repaired later.

Divisions in the palm should be operated upon early by primary end-to-end suture and immobilized for three weeks. Flexor pollicis longus tendon also does well even within its tendon sheath by end-to-end suture, for the functional result is satisfactory. A profundus flexor divided within ½ inch (1.25 cm.) of its insertion can also be sutured and a special technique is described. When both tendons are divided within the digital sheath and when the profundus is severed proximal to ½ inch from its attachment, a free tendon graft gives the only likelihood of a good result. The graft is obtained from a toe extensor, the whole length of the finger is incised laterally and a hole is made in the base of the terminal phalanx. Braided steel wire sutures are recommended. All such operations are done with a tourniquet and are immobilized for three weeks.

Grafts should be done early (the third week is best) and should not be done in the presence of sepsis or digital nerve lesions or in the absence of passive flexion. End-to-end suture of flexor tendons within the tendon sheath does little good and may do harm. Final results should not be evaluated for at least 12 months.

BURNS PLEWES

Transaxillary Approach for Upper Thoracic Sympathetic Ganglionectomy.

W. B. CRANDELL AND W. R. BOSIEN: *Ann. Surg.*, 142: 28, 1955.

The technique of sympathetic denervation of the upper extremity via the axilla is attributed to Goetz of Capetown, followed by Atkins of England. Its more frequent use is advocated, the advantages claimed being the lower morbidity and the greater exposure of the sympathetic chain.

The operation described is through the third interspace in the axilla and across the pleura, retracting the apex of the lung downward. Both the stellate and the second thoracic ganglion are removed. BURNS PLEWES

FORTHCOMING MEETINGS

CANADA

HEALTH LEAGUE OF CANADA, Annual Meeting, Royal York Hotel, Toronto, Ontario. (H.L.C., 111 Avenue Road, Toronto, Ont.) March 5-7, 1956.

CANADIAN TUBERCULOSIS ASSOCIATION, 56th Annual Meeting, Sheraton-Brock Hotel, Niagara Falls, Ontario. (C.T.A., 265 Elgin Street, Ottawa, Ont.) May 15-19, 1956.

CANADIAN PUBLIC HEALTH ASSOCIATION, 44th annual meeting, Admiral Beatty Hotel, Saint John, New Brunswick. (Dr. G. W. O. Moss, Honorary Secretary, 150 College St., Toronto 5, Ont.) May 29-31, 1956.

CANADIAN SOCIETY OF PLASTIC SURGEONS, Annual Meeting, Chantecler Hotel, St. Adele, Quebec. (Secretary-Treasurer, Dr. J. A. Drummond, 1414 Drummond Street, Montreal 25, Que.) June 1-2, 1956.

CANADIAN OPHTHALMOLOGICAL SOCIETY, 19th Annual Meeting, Chateau Frontenac, Quebec, Que. (Dr. R. G. C. Kelly, 90 St. Clair Avenue West, Toronto 7, Ont.) June 7-9, 1956.

CANADIAN UROLOGICAL ASSOCIATION, 12th Annual Meeting, Alpine Inn, Ste. Adele, Quebec (Dr. D. Swartz, Secretary, C.U.A., 332 Medical Arts Building, Winnipeg 1, Man.) June 7-9, 1956.

SOCIETY OF OBSTETRICIANS AND GYNÆCOLOGISTS OF CANADA—1956 Annual Meeting, Manoir Richelieu, Murray Bay, Quebec. (Dr. F. P. McInnis, Secretary, Society of Obstetricians and Gynæcologists of Canada, 1230 Avenue Road, Toronto, Ont.) June 8-10, 1956.

CANADIAN MEDICAL ASSOCIATION, 89th Annual Meeting, Ecole de Commerce, Quebec, Quebec. (Dr. A. D. Kelly, General Secretary, Canadian Medical Association, 150 St. George Street, Toronto 5, Ont.) June 11-15, 1956.

UNITED STATES

AMERICAN ACADEMY OF GENERAL PRACTICE, Eighth Annual Scientific Assembly, Washington. (Mac F. Cahal, A.A.G.P., Broadway at 34 Street, Kansas City 11, Missouri.) March 19-22, 1956.

INTERNATIONAL COLLEGE OF SURGEONS, San Jose, California. (Secretariat, U.S. Section, I.C.S., 1516 North Lake Shore Drive, Chicago 10, Ill.) March 22-23, 1956.

AMERICAN PSYCHOSOMATIC SOCIETY, 13th Annual Meeting, Sheraton-Plaza Hotel, Boston, Massachusetts. (Dr. S. Cobb, Chairman, Programme Committee, 551 Madison Avenue, New York 22, N.Y.) March 24-25, 1956.

THIRD MICROCIRCULATORY CONFERENCE, Hotel Schroeder, Milwaukee, Wisconsin. (Dr. George P. Fulton, Chairman, 1956 Microcirculatory Conference, Department of Biology, Boston University, 657 Commonwealth Avenue, Boston 15, Mass.) April 3, 1956.

INTERNATIONAL ANÆSTHESIA RESEARCH SOCIETY CONGRESS, Flamingo Hotel, Miami Beach, Florida. (Dr. T. H. Seldon, Mayo Clinic, Section on Anæsthesiology, Rochester, Minn.) April 9-12, 1956.

AMERICAN COLLEGE OF ALLERGISTS, 12th Annual Meeting, Hotel New Yorker, New York, N.Y. (Dr. F. W. Wittich, 401 LaSalle Building, Minneapolis 2, Minn.) April 18-20, 1956.

INTERNATIONAL ACADEMY OF PATHOLOGY, 45th Annual Meeting, Cincinnati, Ohio. (Central Office, Armed Forces Institute of Pathology, Seventh Street and Independence Avenue S.W., Washington 25, D.C.) April 24-25, 1956.

AMERICAN GASTROENTEROLOGICAL ASSOCIATION, Annual Meeting, Atlantic City, New Jersey. (The Secretary, A.G.A., University Hospital, Ann Arbor, Michigan.) April 27-28, 1956.

AMERICAN GOITER ASSOCIATION, Drake Hotel, Chicago, Illinois. (Dr. J. C. McClintock, 149½ Washington Avenue, Albany, N.Y.) May 3-5, 1956.

MOUNT SINAI HOSPITAL OF GREATER MIAMI, Sixth Annual Seminar, Fontainebleau Hotel, Miami Beach, Florida. (Dr. Harold Rand, Chairman, 4300 Alton Road, Miami Beach, Fla.) May 17-20, 1956.

NATIONAL TUBERCULOSIS ASSOCIATION: AMERICAN TRUDEAU SOCIETY, Statler Hotel, New York, N.Y. (N.T.A., 1790 Broadway, New York 19, N.Y.) May 20-24, 1956.

CATHOLIC HOSPITAL ASSOCIATION OF THE U.S. AND CANADA, 41st Annual Convention, Milwaukee, Wisconsin. (C.H.A., 1438 South Grand Boulevard, St. Louis 4, Mo.) May 21-24, 1956.

WORLD CONFEDERATION FOR PHYSICAL THERAPY, Second International Congress, New York, N.Y. (Canadian Physiotherapy Association, 8 Bedford Road, Toronto 5, Ont.) June 17-23, 1956.

SECOND INTERNATIONAL CONGRESS ON PHYSIOTHERAPY, New York, N.Y. (Miss M. Elson, American Physical Therapy Association, 1790 Broadway, New York, N.Y.) June 17-23, 1956.

SOCIETY OF NUCLEAR MEDICINE, Hotel Utah, Salt Lake City, Utah. (Secretary, Dr. R. G. Moffat, 2656 Heather Street, Vancouver 9, B.C., Canada.) June 21-23, 1956.

OTHER COUNTRIES

SECOND INTERNATIONAL CONGRESS OF RADIOPHOTOGRAPHY, Paris, France. (Secretariat, S.I.C.R., Via Nazionale 200, Rome, Italy.) April 4-8, 1956.

ASSOCIATION OF CLINICAL PATHOLOGISTS, Cheltenham, England. (Dr. W. H. McMenemey, Maida Vale Hospital for Nervous Diseases, London W. 9, England.) April 7-9, 1956.

FIFTH PAN AMERICAN CONGRESS OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ŒSOPHAGOLOGY, San Juan, Puerto Rico. (Dr. C. E. Munoz MacCormick, Apartado 9111, Santurce 29, Puerto Rico.) April 8-12, 1956.

NATIONAL ASSOCIATION FOR MENTAL HEALTH, Annual Meeting, Harrogate, England. (Miss Applebey, O.B.E., 39 Queen Anne Street, London W.1, England.) April 12-13, 1956.

INTERNATIONAL CONGRESS FOR THE SOCIAL REHABILITATION OF THE LEPER, Rome, Italy. (M. F. Sarsale, International Congress for the Rehabilitation of the Leper, Via Condotti, Palazzo Malta, Rome.) April 16-18, 1956.

ASSOCIATION OF SURGEONS OF GREAT BRITAIN AND IRELAND, Annual Meeting, London, England. (The Secretary, 45 Lincoln's Inn Fields, London W.C.2, England.) April 19-21, 1956.

ROYAL SOCIETY FOR THE PROMOTION OF HEALTH, Annual Congress, Blackpool, England. (Mr. P. Arthur Wells, R.S.P.H., 90 Buckingham Palace Road, London S. W. 1, England.) April 24-27, 1956.

LATIN SOCIETY OF OPHTHALMOLOGY, Second Congress, Madrid, Spain. (Dr. Costi, Montalban 3, Madrid.) April 24-28, 1956.

BRITISH PÆDIATRIC ASSOCIATION, Annual Meeting, Windermere, England. (Dr. P. R. Evans, Institute for Child Health, Great Ormond Street, London W.C. 1, England.) April 25-27, 1956.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM, Annual Congress, London, England. (The Secretary, O.S.U.K., 45 Lincoln's Inn Fields, London, W.C. 2) April 26-28, 1956.

INTERNATIONAL UNION FOR PUBLIC HEALTH EDUCATION, Third Conference, Rome, Italy. (M. Lucien Viborel, Secretary-General, 92 rue St. Denis, Paris Ier, France.) April 27-May 5, 1956.

PROVINCIAL NEWS

MANITOBA

Grandview Hospital No. 3 B will receive from the Manitoba government $15,645, and Crystal City medical nursing unit No. 22 A, $13,878, as interim grants.

Dr. Virginia Apgar, head of the department of anæsthesiology, Presbyterian Hospital, New York, addressed a meeting in the Maternity Pavilion, Winnipeg General Hospital, on January 24 on resuscitation of the newborn.

Dr. A. E. Childe, Winnipeg radiologist, has been elected a Fellow of the American College of Radiology. The investiture was made in Chicago on February 10 at the annual meeting of the College.

The cornerstone of the $750,000 addition to the Medical College was laid in a ceremony on January 19 by the Lieutenant-Governor of Manitoba, the Honourable J. S. McDiarmid. The building was formally opened on the following evening when a convocation of the University of Manitoba was held in the auditorium, which seats about 500. A regular meeting of the Winnipeg Medical Society was also held on the same occasion. The Chancellor of the University, Dr. Victor Sifton, conferred the LL.D. degree, *honoris causa*, on Dr. G. D. W. Cameron, Deputy Minister of National Health, Ottawa, Dr. A. F. Menzies, Morden, and Dr. R. B. Mitchell, Winnipeg. The medical choir, the "Lennox Bell Singers", sang three selections, and Dr. Cameron gave an address on the work of his department with special reference to the control of tuberculosis among Indians and Eskimos. He mentioned the fine work done by several Manitoba medical graduates.

The new building contains the medical library, a bacteriological laboratory, an auditorium and a canteen.

Dr. Percy Johnson, Flin Flon, President of the College of Physicians and Surgeons of Manitoba, presented to the Chancellor a cheque for $11,000 towards the furnishing of the library, and Dr. A. R. Birt, President of the Manitoba Medical Society, gave a cheque for the furnishing of the doctors' reading room.

At the annual banquet of the Morden Chamber of Commerce, Dr. A. F. Menzies was honoured on the occasion of receiving the honorary LL.D. degree from the University of Manitoba. Dr. H. H. Saunderson, President of the University, stressed the importance of the contribution made by university graduates, particularly those in medicine, who are serving in rural Manitoba.

ROSS MITCHELL

SASKATCHEWAN

A postgraduate course on treatment and rehabilitation in chronic illness was held at the University Hospital, Saskatoon, January 12, 13 and 14, sponsored conjointly by the College of Physicians and Surgeons of Saskatchewan, the Medical College, University of Saskatchewan, and the Saskatchewan Public Health Department. Although it was primarily arranged from a medical viewpoint, 165 persons, representing all professional groups concerned in treatment and rehabilitation of the chronically ill and disabled, registered for the three-day course. About one quarter of those attending were practising physicians.

This was the first such programme attempted in the province, and, to our knowledge, in Canada. The guest speakers included Dr. Earl F. Hoerner, Clinical Director, Kessler Institute for Rehabilitation, West Orange, New

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Jersey; Dr. H. Bruce Young, Director, Workmen's Compensation Board Rehabilitation Centre, Malton, Ontario; and Mrs. Betty Ross Hutchison, Head Nurse and Co-Ordinator, Department of Physical Medicine and Rehabilitation, Toronto Western Hospital, Toronto.

The first morning's session, chaired by Dean J. Wendell Macleod, served to keynote the entire course, stressing the need for co-operative team play between the various professional personnel participating in long-term care programmes. The importance of medical leadership was stressed, as was the need for nursing care extending beyond the bedside into re-training for activities of daily living. Caution was directed, however, against developing "factory-like" facilities for the care and rehabilitation of the disabled, in which the personality and needs of the individual patient are neglected. The management of the chronically ill requires, on the other hand, an intense awareness of human emotion and relations. A stimulating interpretation of chronic illness in terms of the need for better understanding of recurrent illnesses and absenteeism given by Dr. I. M. Hilliard completed the session.

The afternoon programme that day was devoted entirely to demonstrations and discussions of chronic children's diseases including cerebral palsy, mental retardation, cœliac disease, scoliosis and a number of orthopædic disabilities.

In the evening, participants were divided into groups according to professional interests, and a number of interesting informal discussions took place.

Clinical demonstrations of rehabilitation techniques took up the entire session on the morning of January 13. These were well received and were carried out by the staffs of the Government Physical Restoration Centres in Saskatoon and Regina, as well as the members of the Department of Rehabilitation Medicine, University Hospital. Problems discussed and demonstrated included poliomyelitis, spinal injuries, paraplegia, quadriplegia, arthritis, hemiplegia and amputations.

A stimulating panel on rehabilitation of severe traumatic problems followed the luncheon, and it, in turn, was succeeded by a symposium on rehabilitation services in Saskatchewan. The majority of those attending evidenced surprise at the magnitude of services potentially available in the province; co-ordination of these services was seen as one of the major steps toward a province-wide programme. (The realization of this essential development led later to the passing of a resolution recommending the establishing of a co-ordinating council to provide an integrated rehabilitation programme in Saskatchewan.)

A banquet that evening, held in the Bessborough Hotel, completed the sessions for the day. Dr. Earl F. Hoerner provided a well-demonstrated and stimulating discussion on upper extremity prosthesis. The dinner concluded with a heartwarming film of a concert pianist's rehabilitation following loss of both arms.

The care of the aged was the theme of the last session on Saturday morning. The magnitude of the problem of caring for our aged population became evident, as did the need for a revision of many previous ideas on the management of elderly patients. A number of housing and institutional care projects was discussed, as were medical and psychiatric problems. Rehabilitation of older patients appeared to be sometimes different from other rehabilitation problems but no less pressing.

The programme's finale was a tour of the various rehabilitation centres in Saskatoon, including the new Department of Rehabilitation Medicine, University Hospital; the Government Physical Restoration Centre; and the Vocational Rehabilitation Centre, operated by the Council for Crippled Children and Adults (Saskatchewan). Those participating in the tour were quite vocal in their praise of the facilities seen.

The Programme Committee, chaired by Dr. T. E. Hunt, Assistant Professor and Chairman, Department of Rehabilitation Medicine, University of Saskatchewan, included Dean J. W. Macleod and Drs. A. E. Buckwold, M. I. Roemer, A. C. Kanaar, and G. W. Peacock, Registrar, College of Physicians and Surgeons of Saskatchewan.

On the whole the response to the course was beyond the expectation of the Committee, and favourable comments have been most gratifying. The only real criticism has been that the programme attempted to cater for too varied a group and that things could only be described summarily. However, the committee, realizing this quite early, had decided that this was the time for a wide orientation course, stressing the essentials of team play and which present-day techniques could be made most useful in the care of the chronically ill and disabled.

The next step would appear, then, to be specific and detailed courses for the individual professions and it is hoped that these can be inaugurated in the near future.

G. W. PEACOCK

NEW BRUNSWICK

At Christmas, Dr. E. A. Petrie, Director of Radiology at St. Joseph's Hospital, Saint John, N.B., was presented with an engraved plaque, to mark the completion of 25 years service in the hospital x-ray department.

The programme of the Saint John Medical Society in January contained a film on "Management of Coronary Artery Disease". This turned out to be a most appropriate subject, because before the film showing the society debated Trans-Canada Medical Plans with special reference to a Canada-wide plan for a great industrial corporation. There was a wide divergence of opinion and wide differences in the quality of rhetoric, but most opinions were expressed with eloquence and considerable heat. Dr. George Keddy, the chairman, proved his ability as a disciplinarian, for the meeting was orderly.

Friends of Dr. H. S. Everett were shocked to hear of the terrible traffic accident early in January which caused the death of Mrs. Everett. Dr. Everett suffered painful injuries but his recovery is progressing well. Dr. Everett and the late Mrs. Everett have for many years taken a very active part in medical affairs in this province, and the sympathy of their many friends is extended to the doctor in his bereavement and suffering.

Interest in the new building programme of the Saint John General Hospital and St. Joseph's Hospital in Saint John is increasing as contractors proceed with additions to both institutions simultaneously. Citizens of the cities of Saint John and Lancaster and indeed of the entire province look forward to the completion of these new services, which will make Saint John an even greater medical centre.

A. S. KIRKLAND

QUEBEC

The Royal Society of Medicine, London, England, announces the award of its triennial Henry Hill Hickman Medal to Dr. Harold Griffith of Montreal, first president of the World Federation of Anæsthesiologists. This is the eighth award of a medal given for original work of outstanding merit in anæsthesia or a directly related subject.

ONTARIO

We are pleased to announce that Mr. Lawrence Plewes, F.R.C.S., surgeon at the Luton and Dunstable Hospital, Bedfordshire, England, the brother of Dr. Burns Plewes of Toronto, is mentioned in the New Year's Honours List. Mr. Plewes has been awarded the C.B.E. for his services to British surgery.

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BOOK REVIEWS

PSYCHOPATHOLOGY AND EDUCATION OF THE BRAIN-INJURED CHILD. Volume II. Progress in Theory and Clinic. A. A. Strauss, President and Director, Cove School for Brain-Injured Children, Racine, Wisconsin and Evanston, Illinois; N. C. Kephart, Professor of Psychology, Purdue University, Lafayette, Indiana, and others. 266 pp. Illust. Grune & Stratton, New York and London; The Ryerson Press, Toronto, 1955. $6.50.

This is the second book in a series planned to deal with the problem of the brain-injured child. The first of this series by Strauss and Lehtinen was in many ways a classic in this field. It was a book which proved of value to physicians, educators, psychologists and almost all those who are interested in the problem of the brain-injured child. The second book in the series is however of a different composition, essentially technical and theoretical. Because of this, it may not appeal to as wide an audience. It will be of greatest value to the psychologist interested in the study of child development and to a lesser degree to the physician and the educationalist.

The various chapters deal with such subjects as embryology and brain injury, psychopathology of perception, psychopathology of language, psychopathology of concept formation and psychopathology of behaviour. There is an excellent chapter dealing with the psychological testing of the brain-injured child of normal intelligence, and also some preliminary conclusions concerning the education of brain-injured children. The final chapter presents a number of case histories of brain-injured children of normal intelligence, outlining the histories, diagnoses, and general programmes of therapy and education.

A DYNAMIC PSYCHOPATHOLOGY OF CHILDHOOD. L. Bender, Professor of Clinical Psychiatry, New York University College of Medicine, New York. 275 pp. Illust. Charles C Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1954. $8.25.

This volume, the third in a series of the Bellevue Studies of Child Psychiatry, is a collection of papers written by Paul Schilder, Lauretta Bender and a number of associates at the Psychiatric Division of the Bellevue Hospital.

The papers deal with facets of the behaviour and experiences of children—symbolism, hallucinations and imaginary companions, notions of what is inside the body, compulsions and obsessions, genital sexual experience, experiences of death of siblings and parents, the meaning of Alice in Wonderland, comic books and children's reactions to war.

The major emphasis of the papers is one that will be welcomed by workers in the mental health field. It consists of attempting to discern how the child orients himself to the world, how he learns what social reality is and the manner in which he may cope with it. Thus the behaviour of the child can only be understood as a continuous process of trial and error which leads to construction and configuration as a basis for action. Behaviour difficulties and neurosis are interruptions of this constructive psychological process.

The essence of the author's approach may be summed up by the word "constructive". Where many psychiatrists and child psychologists have viewed children's fantasy (manifested in hallucinations, imaginary companions and indulging in reading comic books) as unhealthy, regressive or a morbid retreat from reality, Lauretta Bender sees it to be a positive and healthy mechanism called forth during time of need but immediately given up when the child can clearly and spontaneously distinguish from reality itself. Such fantasy can be merely "compensation"—a crutch to help support the weight of threatening social reality—but it can as well be an active and constructive method of exploring possible solutions to problems posed by reality. Bender's follow-up studies tended to belie the contention that such fantasy is necessarily a preliminary symptom of schizophrenia.

The papers, containing many detailed case reports, are interesting and provocative, but in their nature do not furnish an objective basis for acceptance or rejection. The occasional specification of the popular study and the follow-up data are indeed welcome additions to the usual psychiatric document, but they constitute at best a budding rather than a blooming of material that may be assessed objectively.

There is an extensive and well-organized bibliography at the end of this volume which will serve as a source of reference to interested workers.

This volume should prove of value to workers in the field of mental health and especially those in child guidance settings.

CLINICAL ROENTGENOLOGY. VOL. III, THE LUNGS AND THE CARDIOVASCULAR SYSTEM EMPHASIZING DIFFERENTIAL CONSIDERATIONS. A. A. de Lorimier, Radiologist, Saint Francis Memorial Hospital, San Francisco, H. G. Moehring, Radiologist, Duluth Clinic, Duluth, Minnesota, and J. R. Hannan, Radiologist, Cleveland, Ohio. 508 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1955. $22.50.

The third of a four-volume series deals with radiological apects of disease of lungs, heart and other structures of the chest. Clinical findings are correlated and considerable detail of differential diagnosis is discussed. Each of the anatomical structures of the chest is studied separately, and the embryology, roentgen anatomy, and role of various radiological techniques (such as fluoroscopy) are described. Each chapter contains a section entitled "sources of error", in which are discussed many of the radiological features puzzling and difficult for the inexperienced observer to evaluate. The text is very concise and is limited to the essentials, but as a result, descriptions are often too brief and points of diagnostic importance are omitted, while on occasion differential diagnosis is incomplete. The very plentiful illustrations are of excellent quality. A valuable feature is the use of angiocardiograms to demonstrate various features of anatomy of the heart. The bibliography, while adequate, does not appear to contain any references more recent than 1950, which is to be regretted in a field where progress is rapid. (This may account for some of the omissions in the text.) While it does not go into the finer points, this is an excellent general textbook, particularly for physicians interested in chest and heart disease, for those in postgraduate training and for radiologists in the smaller community hospitals.

SEXUAL PRECOCITY. H. Jolly, Consultant Pædiatrician, Plymouth Clinical Area, London, Eng. 276 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1955. $7.50.

This small volume is a welcome addition to the library of the specialist and student. The classification is simple and straightforward. Cerebral causes of sexual precocity are discussed in considerable detail. Perhaps examples of the etiological factors might be more readily impressed on the reader if a typical case was recorded in the text rather than at the back of the book. The caution advised in the use of insufflation techniques for outlining the adrenal is worth noting. The simple explanation with illustrations of the anatomical defect seen in female pseudo-hermaphroditism is excellent. Perhaps the addition of histological sections might be appropriate in the chapter dealing with testicular tumours. The section termed "miscellaneous" is very complete.

This is indeed an excellent, clear and concise review of the subject.

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CARDIAC DIAGNOSIS, A Physiologic Approach. R. F. Rushmer, Associate Professor of Physiology and Biophysics, University of Washington Medical School, Seattle. 477 pp. Illust. W. B. Saunders Company, Philadelphia. 1955. $11.50.

This is a remarkably good book. It is not a textbook of cardiology in the ordinary sense. The physiologic approach is emphasized throughout. Nearly a third of the book is concerned with cardiac physiology based on human and animal experiments. Much of the information is not easily available elsewhere and much has been derived from the author's own research. A careful perusal of these chapters is mandatory for any cardiologist or internist. More than a hundred pages are devoted to a detailed description and evaluation of methods of diagnosis. The diagnosis of cardiac disease occupies the rest of the book and is necessarily somewhat less complete than is found in the larger texts on cardiology. Illustrations are numerous and beautifully presented. This publication can be recommended without reservation.

AIRBORNE CONTAGION AND AIR HYGIENE. An Ecological Study of Droplet Infections. W. F. Wells. 423 pp. Illust. Published for the Commonwealth Fund by Harvard University Press, Cambridge 38, Massachusetts; S. J. Reginald Saunders and Company Limited, Toronto 1, 1955. $6.60.

This book is a compilation of the results of a vast experience in the research and other investigational fields peculiar to the subject. The work is emphatically not one for neophytes. The author has provided a reference work which is unique in its field, and which should have a place in the library of every scientist whose field is in or related to the study of airborne infection and its control, and certainly that of every epidemiologist. It is hardly suitable for general-duties public health personnel.

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THE RELIEF OF SYMPTOMS. W. Modell, Associate Professor, Clinical Pharmacology, Cornell University Medical College, New York. 450 pp. W. B. Saunders Company, Philadelphia and London, 1955. $8.00.

The author has divided his book into three parts. The first section consists of his various arguments for treating the symptoms of disease both in cases where the doctor is attempting to cure the disease and in cases as, for example, of degenerative diseases, where he is seeking to delay the progress of the illness or at least relieve the patient's discomfort. One may not agree with all the author's arguments but at least one must admit that he is very much in earnest about it.

The second part of the book deals with the actual practice of relieving symptoms. This part would be very valuable to any practising physician, as it deals with all the most common symptoms and gives in a most usable form the methods and drugs that are most useful in relieving the various complaints.

The third part reviews the uses, limitations and dangers of ACTH and cortisone.

This book would be a valuable addition to any physician's library.

PAIN. Its Mechanisms and Neurosurgical Control. J. C. White, Chief of Neurosurgical Service, and W. H. Sweet, Associate Visiting Neurosurgeon, Massachusetts General Hospital, Boston. 736 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1955. $19.25.

This book, in the opinion of the reviewer, will long stand as a sign-post on the path of the study of pain, for physician and surgeon as well as neurologist and neurosurgeon. As the title indicates, the mechanism of pain and its neurosurgical control are fully and authoritatively studied, but careful and critical consideration is given to the anatomy of the physiological processes known to be involved in pain. Cobb and Bonner discuss briefly the psychiatric problems involved in the assessment and neurosurgical treatment of pain, including the place of unilateral and bilateral lobotomies.

The book has been published in admirable fashion, and is generously and well illustrated. The comprehensive bibliography is some 35 pages in length. This book from the Neurosurgical Service of the Massachusetts General Hospital is very highly recommended to students and practitioners of medicine seeking help in the understanding and relief of pain.

DENIAL OF ILLNESS. Symbolic and Physiological Aspects. E. A. Weinstein, Associate Attending Neurologist, and R. L. Kahn, Research Psychologist, The Mount Sinai Hospital, New York. 166 pp. Charles C Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1955. $5.25.

This interesting monograph from workers at the Mount Sinai Hospital in New York City will prove of value especially to neurologists, but internists and psychiatrists will also benefit from its study. It is concerned with the symbolic and physiological aspects of *anosognosia*, which the authors regard as a reliable symptom indicating a subcortical lesion in the parietal lobe.

The first chapter deals with historical aspects from the time that the term anosognosia was introduced by Babinski in 1914. The condition had been recognized before this, however, and described, particularly by Head and Holmes in 1911, as a disturbance of the "body scheme".

Of 52 patients who expressed explicit denial of illness, 36 had brain tumours, nine had disease of cerebral vessels, seven subarachnoid bleeding, five lacerating brain injuries, and one meningo-encephalitis, while in one the clinical diagnosis was unclear. (This adds up to 59 patients so it is assumed that some patients had more than one of the above disturbances.) The symptoms, according to the authors, occurred particularly in lesions in the region of the third and lateral ventricles, the diencephalon and midbrain. It did not matter which cortical area was involved by a tumour provided that there was an accompanying involvement of deeper structures.

Many patients with lesions of the parietal area do not show anosognosia in any of the forms described and their "observations indicate that brain damage is not the 'cause' of denial *per se* but rather the level of brain function determines the integration of the *pattern* in which denial is expressed. Thus patients with explicit denial had tended to ignore and rationalize illness and deny felt inadequacies and imperfections long before brain disease developed." Thus they reason that the disability that is denied is not necessarily caused by a lesion in the central nervous system.

From their observations also they state that disturbances in memory found in patients with denial of illness is a selective rather than a general impairment.

This monograph is recommended as a most interesting presentation of a fascinating subject.

SURGICAL PHYSIOLOGY OF THE ADRENAL CORTEX. J. D. Hardy, Surgeon-in-Chief, Hospital of the University of Mississippi, Jackson, Mississippi. 191 pp. Illust. Charles C Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1955. $6.25.

This is a timely monograph of 160 pages dealing succinctly with a complex subject. Dr. Hardy does not attempt to write definitively on his subject—a feat which would be impossible in its present state of flux. He does record systematically what is known of normal and disordered adrenocortical function, discussing the latter successively under: (a) the effects of operation—single and multiple; (b) the effect of burns; (c) adrenocortical function in advanced malignancy and malnutrition; (d) hyperfunction of the adrenal cortex (Cushing's and the adrenogenital syndromes). Finally, therapy is considered under two headings, the first being the place of adrenalectomy in the treatment of essential hypertension and adrenal dependent tumours (breast and prostate), and the second being the use of cortisone and ACTH in a variety of disease processes.

The reviewer found the title incompatible with the content. It is really a broad, shallow review of the whole field without the expected emphasis on surgical problems. As such, however, it is a most useful introduction to one's reading in this field.

CLINICAL PATHOLOGY IN GENERAL PRACTICE. Specially Commissioned Articles from the British Medical Journal, October 1953 to July 1954. 321 pp. Illust. The British Medical Association, Tavistock Square, W.C. 1, London, England, 1955. 21/—.

This book is a collection of articles originally published in the *British Medical Journal* between October 1953 and July 1954. The articles, written by a variety of authors of note, have been fully revised for publication in book form. Although primarily written for the general practitioner working under the facilities of the British national health and laboratory services, this book is a most valuable guide for practitioners anywhere.

Without going into technical details it gives pertinent information about all phases of laboratory work, which can be of help to the general practitioner, and will increase his diagnostic acumen. Too often these days, one hears the complaint that the general practitioner has become a clearing house, and that specialization has removed interest and glamour from his daily work. This book certainly points out how much can be done and should be done by the general physician to arrive at a diagnosis, before he has to resort to the services of a specialist. It is also refreshing to read about limitations of the value of certain tests in this book. Generally speaking, it will be a most valuable addition to any physician's library.

Canad. M. A. J.
Mar. 1, 1956, vol. 74

PHARMACOPŒA INTERNATIONALIS, INTERNATIONAL PHARMACOPŒIA. 1st ed. Volumes I and II, 350 pp. World Health Organization, Palais des Nations, Geneva, 1955. $6.75.

In May 1950 the Third World Health Assembly approved the publication of an international pharmacopœia, recommending in accordance with one of the Articles of the WHO Constitution the eventual inclusion of its provisions in national pharmacopœias. Volume I, presented at the 14th General Assembly of the International Pharmaceutical Federation in Rome in September 1951, was favourably received and is now widely used. Any Member State of the World Health Organization may include part or all of the provisions of the work in its national pharmacopœia.

The urgent need for the publication of Volume I to assist in co-ordinating and unifying the health activities of the Member States made it necessary to omit certain drugs in which rapid advances in knowledge and practice were being made. Volume II supplies these omissions and deals with various forms of insulin, with tubocurarine chloride and with antibiotics: chloramphenicol, chlortetracycline, dihydrostreptomycin, oxytetracycline, penicillin, and streptomycin. Synthetic chemical drugs recently introduced are included and an attempt is made to complete the information given in Volume I by including monographs on compressed tablets, sterile injections, and tinctures.

A Table of Unusual and Maximal Doses of the substances described in the volume is included, and a Table of Usual Daily Doses for Children covers the range of drugs in both Volumes I and II which are in general use in pædiatrics. The doses stated are adjusted in accordance with the sensitivity of children and are not calculated from adult doses by definite rule; they are given as general indications for modification by the physician in accordance with his judgment. In the compilation of these tables much assistance was given by the World Medical Association.

A section on solutions of cardiolipin and lecithin for serological tests has been included at the request of the Subcommittee on Serology and Laboratory Aspects of the Expert Committee on Venereal Infections and Treponematoses and was prepared with their collaboration. Although this section is outside the usual scope of a pharmacopœia, it was included to provide in readily accessible form the information necessary for the control of these substances.

Among the appendices is a list of international biological standards, approved by the Expert Committee on Biological Standardization.

Volume II was prepared by members of the World Health Organization's Expert Advisory Panel on the International Pharmacopœia and Pharmaceutical Preparations and by the WHO Secretariat. A general revision of Volumes I and II is now under way. Opinions on the subject matter are invited, and comments and suggestions will be considered for possible integration into subsequent printings.

BASIC SURGICAL SKILLS. A Manual with Appropriate Exercises. R. Tauber, Assistant Professor of Gynecology and Obstetrics, The Graduate School of Medicine, University of Pennsylvania, Philadelphia. 75 pp. Illust. W. B. Saunders Company, Philadelphia, 1955. $3.75.

This book outlines and illustrates a number of the common ligating and suturing techniques used in surgery. It also describes a board set-up on which these manœuvres can be practised. Methods of obtaining sure hæmostasis are discussed. Those beginning a surgical internship or residency will find its perusal worth while.

Books Received

Postgraduate Obstetrics and Gynæcology, 2nd Ed. F. J. Browne, Emeritus Professor of Obstetrics and Gynæcology, and J. C. McClure Browne, Professor of Obstetrics and Gynæcology, London University, England. 725 pp. Illust. Butterworth & Co., Ltd., London, 1955. $14.00.

Corneal Grafts. B. W. Rycroft, The Corneo-Plastic Unit and Eye Bank, Queen Victoria Hospital, East Grinstead, Sussex, England. 296 pp. Illust. Butterworth & Co., Ltd., London, 1955. $11.50.

The Body Fuids. J. R. Elkington, Associate Professor of Medicine, University of Pennsylvania, and T. S. Danowski, Renziehausen Professor of Research Medicine, University of Pittsburgh. 626 pp. Illust. Williams & Wilkins Company, Baltimore; Burns and MacEachern, Toronto, 1955. $10.00.

Fractures of the Facial Skeleton. N. L. Rowe, Consultant in Oral Surgery, Plastic and Maxillo-facial Surgery Unit, and H. C. Killey, Consultant in Oral Surgery, Plastic and Maxillo-facial Surgery, Rooksdown House, Basingstoke, Hants., England. 923 pp. Illust. E. & S. Livingstone, Ltd., Edinburgh and London; The Macmillan Company of Canada, Ltd., Toronto, 1955. $20.50.

A Psychiatrist Looks at Tuberculosis. Eric Wittkower, Associate Professor of Psychiatry, McGill University, Montreal. 164 pp. The National Association for the Prevention of Tuberculosis, London, 1955. $3.00.

Doctor at Large. Richard Gordon. 208 pp. Michael Joseph Limited, London, 1955. $2.50.

Self-Help for the Arthritic. David White, Senior Psychotherapist, the Walnut Tree Hospital, Sudbury, England. 55 pp. Illust. Henry Kimpton, London, 1955. $.75.

Community Programs for Mental Health. Edited by Ruth Kotinsky and H. L. Witmer. 362 pp. Harvard University Press; S. J. Reginald Saunders and Company Limited, Toronto, 1955. $5.50.

James Parkinson 1755-1824. Edited by M. Critchley. 268 pp. Illust. The Macmillan Company of Canada Limited, Toronto, 1955. $2.50.

Biologie des Maladies dues aux Anaerobies. (Biology of Diseases due to Anaerobes). A. R. Prevot, Institut Pasteur, Paris. 572 pp. Editions Médicales Flammarion, Paris, 1955. Fr. 2,225.

Cybernetics. Circular Causal and Feedback Mechanisms in Biological and Social Systems. Transactions of the 10th Conference April 22, 23 and 24, 1953. Edited by H. von Foerster, Professor of Electrical Engineering, University of Illinois, Chicago, 100 pp. The Josiah Macy Jr. Foundation, New York, 1955. $2.75.

Chlorpromazine and Mental Health. Proceedings of the Symposium held under the auspices of Smith, Kline & French Laboratories June 6, 1955, Warwick Hotel, Philadelphia. 200 pp. Lea and Febiger, Philadelphia, 1955; The Macmillan Company of Canada, Toronto, 1955. $3.00.

Amputees and Prostheses. Report of a Conference on Prosthetics, Copenhagen, 23-28 August, 1954. 52 pp. World Health Organization Technical Report Series, No. 100, World Health Organization, Palais des Nations, Geneva, 1955. $.60.

The Diagnosis and Management of Urological Cases. B. W. T. Pender, Senior Surgical Registrar, St. George's Hospital, and J. O. Robinson, Senior Surgical Registrar, St. Bartholomew's Hospital, London. 170 pp. Illust. Baillière, Tindall and Cox, London; The Macmillan Company of Canada, Toronto, 1955. $3.60.

Control of Insect Vectors in International Air Traffic. A Survey of Existing Legislation. 59 pp. World Health Organization, Palais des Nations, Geneva, 1955.

Bulletin of the World Health Organization. Vol. 13, No. 4. 742 pp. Illust. World Health Organization, Palais des Nations, Geneva, 1955. $2.00.

JOURNAL OF

Canadian Medical Association

Editorial Office—150 St. George St., Toronto 5

General Secretary's Office—150 St. George St., Toronto 5

SUBSCRIPTION RATES

The Journal is supplied to paid-up members of the Canadian Medical Association as a perquisite of membership. Medical libraries, hospitals, and individuals may subscribe to the Journal at $12.00 a year, payable in advance. There is a special rate for medical students residing in Canada of $2.50 a year. Subscriptions and all relative correspondence should be addressed to the office of the Managing Editor at 150 St. George Street, Toronto 5, Ontario.

INSTRUCTIONS TO CONTRIBUTORS

Manuscripts: Manuscripts of original articles, case reports, clinical and laboratory notes, and special articles should be submitted to the Editor at the C.M.A.J. editorial office, 150 St. George St., Toronto, with a covering letter requesting consideration for publication in the *Journal*. Acceptance is subject to the understanding that they are submitted solely to this *Journal*, and will not be reprinted without the consent of both the Editor and the author. Articles should be typed on one side only of unruled paper, double-spaced and with wide margins. Carbon copies cannot be accepted. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents.

The Editor reserves the right to make the usual editorial changes in manuscripts; these include such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity to *Journal* style. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints may be ordered on a form supplied with galley proofs.

References: Authors should limit references to published work to the minimum necessary for guidance to readers wishing to study the subject further. They should not quote articles they have never seen, and should set out references in a numbered list at the end of the article, thus:

1. Doakes, J.: *M. J. Kamchatka*, 1: 2, 1955, giving in order: (1) Author's name and initials in capitals. Where more than three authors are concerned in an article, only the first should be named, with *et al.* as reference to the others. (2) Cumulative Index Medicus abbreviation of journal name. (3) Volume number. (4) Page number. (5) Year.

References to books should be set out as follows:

Pickwick, S., *Textbook of Medicine*, Jones and Jones, London, 1st ed., p. 30, 1955.

Illustrations: Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10 by 8 inches. Colour work can be published only at the author's expense. Magnification of photomicrographs must always be given. Photographs must not be written on or typed on. Identification can be made by pasting an identifying legend on the back. Patients must not be recognizable in illustrations, unless the written consent of the subject to publication has been obtained. Graphs and diagrams should be drawn in india ink on suitable white paper. Legends to all illustrations should be typed separately from the text of the article. Illustrations should not be rolled or folded.

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Please send copy to Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

Rates: $5.00 for each insertion of 40 words or less, additional words 10c each.

If a box number is requested, there will be an additional charge of 50c on the first advertisement to cover postage and handling charges.

Miscellaneous

NOTICE.—Going overseas this year? You can save money by buying your foreign currency requirements in Canada before your departure. Rates are even more favourable now than later in the travelling season. Free information from Deak & Co., Inc., Foreign Exchange brokers, 77 Adelaide Street West, Toronto. Telephone EMpire 6-4461.

FOR SALE.—Sanborn Viso-cardiette (almost new), Wapler cautery, I new colorimeter, Leitz (Westinghouse), Clay-Adams enclosed centrifuge—4 tube head; 1 B & L microscope with three objectives, bell jar and case; and 1 Prometheus chrome sterilizer. Apply to Box 627, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

Office Space

FOR RENT.—Doctor's office and waiting room. Excellent location in West End of Toronto. Available July 1, 1956. Apply (Toronto) ROger 7-3300.

OFFICE SPACE FOR RENT.—Ville St. Laurent, Montreal suburb. Approximately 600 feet, modern new building, first floor, doctors and dentists only. Reasonabe rent. Best location in St. Laurent. Population approximately 40,000, half are children under sixteen years of age. Dec-Mac Corporation, 1470 Peel Street, Montreal, Quebec.

Positions Vacant

RADIOLOGIST WANTED for holidays, during the months of July or August, in pleasant lake district city. Car is necessary. May be possible to provide accommodation. Adequate remuneration provided. Could also use help one-two weeks in March or April. Reply to Box 632, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

ASSISTANT WANTED.—Recent graduate wanted for growing small town practice in Southern Ontario. Excellent nearby hospitals; organized hours. Opportunity for partnership. Please state qualifications, religion, race, telephone number, etc., to Box 633, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED.—Locum tenens for one year for practice in Dawson City. To commence not later than June 1, 1956. Completely furnished and heated house, and car expenses. Salary according to qualifications. Apply to Dr. J. H. Rooks, Box 508, Dawson City, Y.T.

TWO ASSISTANTS in general practice required for Northern Ontario clinic, beginning July 1, 1956. Reply to Box 584, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED.—Young general practitioner with some anæsthetic experience for Southern Saskatchewan town with good hospital. Initially assistant with view to partnership. Reply to Box 612, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

POSITION VACANT.—Certified pathologist (or the equivalent) wanted to join the staff of a large Canadian general hospital in a university centre. General training in both pathological anatomy and clinical pathology are essential. For further information apply to Box 609, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED. — ASSOCIATE PATHOLOGIST. — Certified or eligible for St. Lawrence County, New York State. Salary $12,500-$15,000, depending on experience. United States citizenship not required; eligibility for New York State license necessary. Write Dr. Robert T. Rogers, Director, St. Lawrence County Laboratories, Canton, New York.

WANTED.—Young general practitioner for small group in mid-western Canadian city. Salary dependent on qualifications. Eventual partnership anticipated. Reply to Box 575, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

INTERNIST WANTED with certification or fellowship for an expanding group practice in Ontario. Some experience in neurology and/or psychiatry would be helpful. Apply stating experience, qualifications, starting salary expected, and include a recent snapshot if possible. Box 585, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

CLASSIFIED ADVERTISEMENTS

WANTED. — ANÆSTHETIST. — Well established group in central Ontario requires services of certified anæsthetist. Write stating remuneration requirements, experience and references to Box 562, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED.—Urologist with certification or fellowship for a group in Ontario. Apply stating experience, qualifications, starting salary expected, and include a recent snapshot if possible. Box 540, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

ASSISTANT required for busy general practitioner in suburban Toronto area. Opportunity for partnership. Please write giving age, experience and religion to Box 466, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED BY GROUP in Central Ontario, recent graduate, with one year's internship, who wishes employment for one year before specializing. Adequate remuneration, car allowance, rotating duty schedule, etc. Apply giving pertinent information to Box 595, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED.—Physician, general practice for City of Hope, North Dakota, and vicinity. New medical building and ambulance service available. Excellent opportunity for doctors. Send inquiries to Hope Clinic Board, Hope, North Dakota, U.S.A.

WANTED.—Internist with fellowship for progressive Ontario clinic. Reply stating age, previous experience in practice, and salary expected. Box 586, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED.—Assistant in busy general practice in English-speaking Montreal suburb; beginning summer 1956; prospect of partnership. State age, qualifications, race, religion and special interests. Box 508, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

POSITION VACANT.—A vacancy exists on the active staff of this English-speaking general hospital for a doctor with certification in E.N.T. or E.E.N.T. to take charge of the department. All tonsillectomies referred to the E.N.T. department. A new hospital on a new site is under construction for completion in 1956. For further details apply to: Administrator, Jeffery Hale's Hospital, Quebec City, P.Q.

WANTED.—LOCUM TENENS for months of July and August in Ottawa valley rural practice. $450 per month, car provided and car allowance. Also full board for single man. Reply stating age, experience, religion and marital status, to Box 631, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

PHYSICIAN WANTED for general practice in group. Modern, up-to-date offices with all facilities. Particularly attractive contract. Please reply, stating training and salary expected, to Box 628, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

PSYCHIATRISTS. — Kansas license or fellowship. Board certified or board eligible. Salary range: $9,456 to $13,226 per year plus partial maintenance, depending upon the qualifications of the individual. Apply to Superintendent, Larned State Hospital, Larned, Kansas, U.S.A.

PSYCHIATRIC HOSPITAL CLINICAL DIRECTOR.—Requirements: Rotating internship in an approved hospital. Certified by A.P.A. Salary range: $12,000 to $15,312 per year plus partial maintenance. Apply to Superintendent, Larned State Hospital, Larned, Kansas, U.S.A.

POSITIONS VACANT.—Resident and senior assistant residents in medicine appointments, University Alberta Hospital, Edmonton, Alberta, commencing July 1, 1956. Apply to Assistant Medical Superintendent.

GENERAL PRACTICE RESIDENTS — FOR FULLY APPROVED AND ACCREDITED 125-BED GENERAL HOSPITAL WITH LARGE OUT-PATIENT CLINIC. ACTIVE TEACHING PROGRAMME BY STAFF OF CERTIFIED SPECIALISTS. GUARANTEED PRACTICE OPPORTUNITY ON COMPLETION OF TRAINING. SALARY $5,000 PER YEAR. CONTACT: DIRECTOR OF MEDICAL EDUCATION, THE LYNN HOSPITAL, DETROIT 17, MICHIGAN, U.S.A.

WANTED.—Certified obstetrician and gynæcologist to join group in Alberta. Reply to Box 623, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

UNIVERSITY OF ALBERTA HOSPITAL. — Fellowships in medicine and surgery. Two fellowships in the department of medicine and one in the department of surgery are open to suitably qualified postgraduate students wishing to take one year in clinical investigation. Salary $3,000 per annum. Applicants interested in these posts should apply to Dr. A. C. McGugan, Superintendent of the University of Alberta Hospital, Edmonton, Alberta, with a curriculum vitæ and two letters of reference. Further details will be supplied to interested candidates.

Continued on Page 36

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

INTENSIVE POSTGRADUATE COURSES

STARTING DATES—SPRING 1956

SURGERY—Surgical Technic, Two Weeks, April 2, April 16.
Surgical Anatomy and Clinical Surgery, Two Weeks, June 18.
Surgery of Colon and Rectum, One Week, April 9, May 7.
General Surgery, Two Weeks, April 23.
Basic Principles in General Surgery, Two Weeks, April 9.
Thoracic Surgery, One Week, June 4.
Esophageal Surgery, One Week, June 11.
Breast and Thyroid Surgery, One Week, June 18.
Gallbladder Surgery, Ten Hours, April 9, June 25.
Fractures and Traumatic Surgery, Two Weeks, June 18.
Varicose Veins, Ten Hours, April 30, June 18.

GYNECOLOGY—Office and Operative Gynecology, Two Weeks, April 16, June 18.
Vaginal Approach to Pelvic Surgery, One Week, April 30, June 11.

OBSTETRICS—General and Surgical Obstetrics, Two Weeks, May 7.

MEDICINE—Internal Medicine, Two Weeks, May 7.
Electrocardiography and Heart Disease, Two Week Basic Course, March 12.
Gastroenterology, Two Weeks, April 23.
Dermatology, Two Weeks, May 7.

RADIOLOGY—Diagnostic X-Ray, Two Weeks, April 30.
Clinical Uses of Radioisotopes, Two Weeks, May 7.

PEDIATRICS—Intensive Review Course, Two Weeks, May 14.
Neurological Diseases: Cerebral Palsy, Two Weeks, June 18.

UROLOGY—Two-Week Course April 16.
Cystoscopy, Ten days, by appointment.

TEACHING FACULTY—ATTENDING STAFF
OF COOK COUNTY HOSPITAL

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Department of National Health and Welfare
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For details, please write IMMEDIATELY to:
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Quote Competition 56-484.

CLASSIFIED ADVERTISEMENTS

LOCUM TENENS WANTED for general practice in Northwestern Ontario for 2 or 3 months between June and September of this summer. 22-bed modern hospital. Prefer at least 1 year rotating internship in Grade A hospital. Salary $600 to $700 per month with car allowance. Apply to Doctor A. H. Jeffrey, Box 250, Nipigon, Ontario.

WANTED.—An assistant with a view to partnership for a busy general practitioner in a Western Ontario town, with own hospital facilities. Opportunity to net at least $10,000 the first year. Please reply stating age, experience, marital status and religion to Box 621, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

RADIOLOGIST WANTED for 60-bed general-public hospital, within one hour of metropolitan Toronto. Send full particulars, remuneration required, etc., to Box 618, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

APPLICATIONS are invited for the post of assistant professor of bacteriology. Initial salary $5,000. Superannuation benefits provided. Duties commence July 1, 1956. Letters of application with details of qualifications and experience and names of two referees should be submitted not later than March 31, 1956, to Dr. J. W. Macleod, Dean of Medicine, University of Saskatchewan, Saskatoon, Saskatchewan.

CERTIFIED OBSTETRICIAN wanted for group practice in Western Ontario town. Permanent position if satisfactory. Reply giving references and stating salary expected to Box 596, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED FOR GENERAL PRACTICE.—Qualified general practitioner with special interest in obstetrics and pædiatrics. Only those seeking permanent position need apply. Remuneration commensurate with training and ability. Apply Box 617, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

BUSY ONTARIO GENERAL PRACTITIONER wishes assistant with working knowledge of anæsthesia, surgery and obstetrics. Remuneration will depend on ability and willingness to work. Open hospitals. Permanent position to right man. Full particulars in first letter please. Box 615, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

AVAILABLE ASSISTANTSHIP for large medical and surgical practice with general practitioner in Southern Ontario. Good hospital, good wages, and liberal car allowance. Varied and full general practice. Young Canadian graduate preferred. The above position open immediately for terms of one month up to an indefinite period. Similar position for a term of one year available July 1. Apply Box 614, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

OPPORTUNITIES IN THE ONTARIO MENTAL HEALTH SERVICE.—A training programme leading to eligibility for certification by examination in the specialty of psychiatry by the Royal College of Physicians and Surgeons (Canada) is offered while serving in the Ontario Hospital Service. Applicants are required to have completed at least a one-year rotating internship in an approved hospital. They must be in possession of a license to practice medicine in the province of Ontario. The starting salary is $4,500 per annum, plus a cost of living bonus of $120 per annum, with annual increments for satisfactory service. During the first four years a physician in the Ontario Hospital Service is under training. The first year is usually spent in one of the Ontario hospitals. The second and third years are usually spent at the Toronto Psychiatric Hospital where a diploma course in psychiatric medicine is offered by the University of Toronto. Also, at the University of Western Ontario there is a similar training programme of two years centring around the medical school. On successful completion of the university course and transfer back to an Ontario hospital, physicians, on recommendation, are promoted to a minimum salary of $6,500 per annum, plus the $120 cost of living bonus. The fourth year is usually spent at an Ontario hospital approved for training specialists in the field of psychiatry. At the end of four years of the training programme, physicians are expected to sit for examinations for the Specialist Certificate in Psychiatry from the Royal College of Physicians and Surgeons (Canada). After obtaining certification as a specialist, physicians, if recommended, automatically go to a minimum of $7,500 per annum, plus the $120 cost of living bonus. Increases beyond this figure are given by annual increments or promotion to higher positions. Following certification as a specialist, a fairly wide variety of positions are available for physicians as senior staff psychiatrists on hospital duty, in charge of mental health clinics, or in charge of a community psychiatric clinic. Further information may be obtained by writing to: Chief, Mental Health Division, Ontario Department of Health, Parliament Buildings, Toronto, Ontario.

Positions Wanted

ENGLISH GRADUATE, age 29, married, two children, seeks opening in general practice, either single or with group in Prairie Provinces. Residencies in internal medicine, midwifery, anæsthetics. 8 months' experience in general practice. Reply to Dr. Charles F. Wood, Radville, Saskatchewan.

CLASSIFIED ADVERTISEMENTS

GENERAL SURGEON, Canadian graduate, age 33, married, two children, completing 4 years' approved surgical residency July 1956. Prefer association with individual or group. Highest character and professional references. Reply to Box 613, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED.—Recent Canadian graduate, with two years' postgraduate medicine, desires practice either with a group, an experienced practitioner, or will take over from an M.D. leaving a practice. Preferably in Ontario or the Maritimes. Married. Protestant. Reply to Box 608, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED: partnership or assistantship in general practice by young Irish graduate completing senior intern year July. Writing L.M.C.C. May. Interested particularly in obstetrics and gynæcology. Western provinces preferred. Box 605, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

LONDON GRADUATE, age 31, married, seeks E.N.T. position with group or clinic. Qualified M.B., B.S., 1948. Usual resident appointments. 5 years' E.N.T. experience. Diploma in 1953. Reply to Dr. Alan G. McCallum, 51 Cranmore Lane, Aldershot, Hants, England.

SURGEON, M.D. (Cambridge), F.R.C.S. (England), 34, Briton; special interest and experience in accident surgery—England and U.S.A.; seeks similar work in Canada. Currently employed in English teaching hospital. Reply to A. F. Care, 516, 22nd Avenue S. W., Calgary, Alberta.

SCOTSMAN, EDINBURGH GRADUATE, married, arriving in April, seeks entry into practice in Alberta. Hospital and outside experience in Britain and New Zealand. Interests: surgery, obstetrics, anæsthetics. Wishing to settle. Please airmail replies to Dr. McIntyre, c/o Munro, 19, Landscape Road, Papatoetoe, Auckland, New Zealand.

CERTIFIED, 1955, general surgeon, age 30, desires position with group, clinic or surgeon, anywhere in Canada, preferably Ontario. Reply to Box 625, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

TWO DOCTORS, one F.R.C.S. (Edinburgh) and the other D.R.C.O.G. (London), seek suitable location to engage in general practice in partnership. Willing to buy if necessary. Reply to Box 622, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

LOCUM TENENS WANTED by McGill graduate for all or part of April, May, June, 1956. Experienced in general practice, licensed in Ontario. Training—1 year rotating internship plus 3 years' surgery. References furnished. Reply to Box 620, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

ANÆSTHETIST, experienced in all types of modern anæsthesia except cardiac surgery, would like post with group or clinic. 10 years' general practice before specializing. Married, 2 children. Available early April. Reply to Box 619, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

ENGLISH SURGEON, F.R.C.S., L.M.C.C., late colonial medical service, age 43, single; wide experience in general and traumatic surgery, urology and E.N.T.; desires association with group or private practice, willing to do some general practice. Southern Ontario preferred. Reply to Box 616, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

WANTED: SURGICAL PRACTICE.—Canadian, with family; recent graduate; 4 years' surgical residency to be completed in July. To associate with fellow, or practice alone. Prefer Eastern Canada, but not exclusively. Reply to Box 630, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

McGILL GRADUATE, currently in United States, desires return to Canada, preferably Montreal area. Certified by American Board of Internal Medicine, licensed as specialist by Quebec Board, associate American College of Physicians. Desires association with established internist or general practitioner, membership in group, part-time industrial position, or take over practice of retiring doctor. Available July 1956. Reply to Box 545, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

Practices

UNOPPOSED RURAL SASKATCHEWAN medical and surgical practice, 13-bed union hospital; no contracts; all fee for service. Fully modern home including all furniture (television, electric dryer, stove, fridge, etc.). Total cost $11,500. Some terms. Incumbent leaving for Quebec. Available June 1956, or sooner if desired. Reply to Box 598, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

FOR SALE.—Saskatchewan. Active general practice in prosperous rural area. Modern hospital facilities. One other doctor practising in district. Modern house available. Owner specializing. Terms can be arranged. Reply to Box 629, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

(Continued on page 38)

CLASSIFIED ADVERTISEMENTS

TO RENT.—Doctor's office, with dispensary; separate oil hotwater heating; private entrance, records. Patients await successor In beautiful south-western Ontario town of 6,000. Apply to Box 626, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

FOR SALE.—Toronto, central location. Radiological practice, completely equipped. Almost new 500 M.A., G.E. make. Suitable for radiologist or for hospital installation. Many extras. Price new was well over $28,000. Will sell for cash or cash deposit plus monthly payments. Reply to Box 624, Canadian Medical Association Journal, 150 St. George Street, Toronto 5, Ontario.

Internships and Residencies

RESIDENCY IN PÆDIATRICS.—There is an opening for one resident, July 1, 1956 to June 30, 1957 in pædiatrics at the Royal Alexandra Hospital. Salary $175 per month plus maintenance and laundry. Service covers isolation, maternity and pædiatrics, or 200 beds in all. Applications should be made to Dr. D. R. Easton, Medical Superintendent, Royal Alexandra Hospital, Edmonton, Alberta.

PATHOLOGY RESIDENCIES AND FELLOWSHIPS available commencing June 1, 1956 at the Winnipeg General Hospital. Approved for training by the Royal College of Physicians and Surgeons and for American Boards. Apply to: The Director, Department of Pathology, Winnipeg General Hospital, Winnipeg, Manitoba.

RESIDENCY IN MEDICINE.—There is an opening for one resident, July 1, 1956 to June 30, 1957 in medicine at the Royal Alexandra Hospital. Salary $175 per month, plus maintenance and laundry. Applications should be made to Dr. D. R. Easton, Medical Superintendent, Royal Alexandra Hospital, Edmonton, Alberta.

ST. LUKE HOSPITAL in Montreal, capacity of 451 beds, is considering applications for internship or residencies in the different services of a general hospital and most specially in the following services where the teaching is approved by the American College of Surgeons: surgery, medicine, obstetrics, oto-rhinolaryngo-ophthalmology, pathology and radiology. Applicants may address their applications to Doctor H. I. Tétreault, Medical Superintendent.

PSYCHIATRIC RESIDENCIES.—HOSPITAL WITH LARGE MEDICAL STAFF OFFERS FULLY ACCREDITED THREE YEAR TRAINING PROGRAMME BEGINNING JULY 1, 1956 FOR MEN AND WOMEN DESIRING CERTIFICATION IN PSYCHIATRY. INCLUDES UNIVERSITY POSTGRADUATE COURSE, GUEST LECTURES, TRAINING IN MODERN THERAPEUTIC PROCEDURES AND SUPERVISED WORK IN MENTAL HYGIENE CLINICS. INITIAL SALARY INCLUDES FULL FAMILY MAINTENANCE. REPLY TO BOX 606, CANADIAN MEDICAL ASSOCIATION JOURNAL, 150 ST. GEORGE STREET, TORONTO 5, ONTARIO.

POSITION VACANT.—Available 1 year senior internship in pathology. $150 per month, room, board, etc. University affiliation. Apply to Pathologist, Hotel Dieu Hospital, Kingston, Ontario.

APPROVED ROTATING INTERNSHIPS AND RESIDENCIES in medicine, neurology, psychiatry. 684-bed county hospital near New York city. Exceptional educational opportunity. Apply: Bergen Pines County Hospital, Paramus, New Jersey. Interns: Only applicants of approved medical schools will be considered. Stipend $100 monthly plus complete maintenance. Residents: Applicants must have completed one-year approved internships. Stipend $200 monthly plus complete maintenance.

AVAILABLE IMMEDIATELY.—Obstetrician and gynæcologist assistant residency, one year approval. 224-bed general hospital, modern, well equipped; intern and resident programme. House staff allowed full range under proper medical supervision. Full maintenance and uniforms. Monthly stipend $250. Class A medical school graduates only. The Lawrence and Memorial Associated Hospitals, New London, Connecticut. William J. Murray, Jr., M.D., Chairman, Committee on Residents and Interns.

TWO PÆDIATRIC RESIDENTS required for a period of one year, commencing July 1, 1956, for 83-bed children's acute and long-term medical and surgical hospital. University teaching programme. Accommodation available. Remuneration $175 per month, less $15 per month accommodation. Meals available if required. Application stating age, qualifications, nationality, marital status, accompanied by recent references and photograph should be forwarded by airmail to: The Administrator, Children's Hospital, 250 West 59th Avenue, Vancouver 15, British Columbia.

APPLICATIONS WILL BE RECEIVED for the following positions, duties to begin July 1, 1956. Medicine: 1 assistant resident; 2 senior internships. Surgery: 1 senior internship. Pædiatrics: 1 assistant resident. Pathology: 1 assistant resident; 2 senior internships. Rotating: 3 rotating senior internships. Apply in writing to: Doctor P. L'Heureux, Medical Director, St. Boniface Hospital, St. Boniface, Manitoba.

NEWS AND NOTES

VOLUNTARY MEDICAL AND HOSPITAL INSURANCE*

This is a follow-up and extended study of the voluntary medical care and hospital plans of Canada, including non-profit and private insurance schemes. It is complementary to the memorandum on public hospital and medical care plans. As regards hospitalization, voluntary plans take care of about 40% of the population of Canada, while compulsory plans cover another 20%. When to this is added the coverage of other groups such as Indians, Eskimos, veterans and servicemen, it is clear that a substantial majority of Canadians have some insurance against the growing costs of hospital care. The proportion of the population insured for other medical care is of course less but has grown from 10% to 30% in the five years ending January 1954.

Like its companion volume on public hospital and medical care plans, this is essential reading for all interested in medical economics, and desirable reading for all practising physicians.

*Voluntary Medical and Hospital Insurance in Canada General Series, Memorandum No. 9. Research Division, Department of National Health and Welfare, Ottawa.

FEDERAL HEALTH GRANTS

In Manitoba a grant of $215,848 goes towards construction of a new unit for female patients at the Manitoba School for the Mentally Defective, Portage la Prairie. Features of the new structure will include, in addition to accommodation for 159 patients, day rooms, physiotherapy facilities, operating room, laboratory, and dental and x-ray facilities.

In Alberta a grant of $70,996 will help meet construction costs of the new addition to the provincial mental hospital, Ponoka, with accommodation for 93 more patients.

MENTAL HEALTH STATISTICS 1954

The mental health statistics published by the Dominion Bureau of Statistics for Canada in 1954 reveal the highest rate of hospitalization for mental illness yet reported. The number of patients in residence in mental hospitals and psychiatric wards at the end of 1954 represented a rate of 410.9 per 100,000 population. Since there was a bed capacity of only 358.3 per 100,000, it is clear that there was a considerable overcrowding in spite of the substantial increase in the number of available beds. In fact over 62,000 patients were being accommodated in wards with a combined capacity of only 54,346 beds.

The admission rate (first admissions plus re-admissions) of 193.5 was the highest ever, but this was to some extent offset by the increased discharge rate, also a record. It must not be thought, however, that all those discharged were greatly improved. Only 24.2% of discharges were recovered or "much improved" although 84.4% were sent home; hence at least 60% of all discharges (10,000 persons) reentered the community with some considerable amount of psychiatric

(Continued on page 43)

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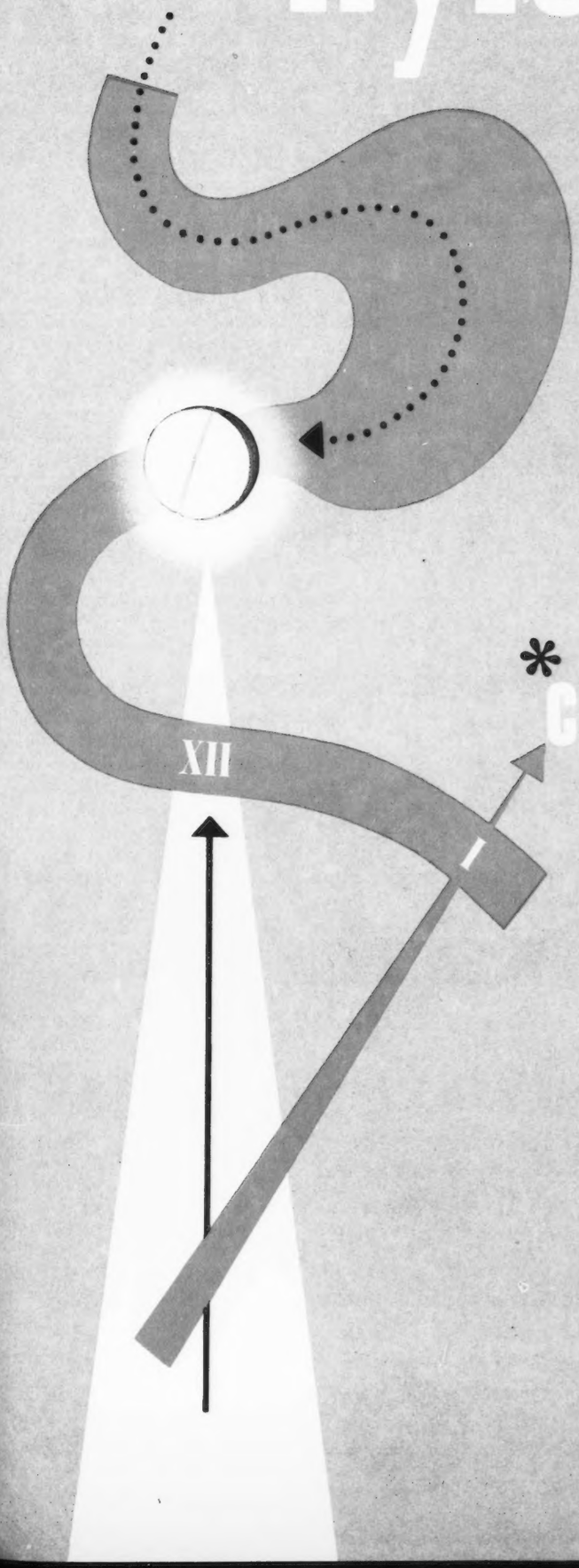

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In 267 patients,* postpartum breast engorgement and lactation were suppressed in 96.2 per cent, without nausea, vomiting, breast abscess, excessive lochia, withdrawal bleeding, or virilization. One particular advantage noted was the absence of mental depression, which usually occurs about the fourth day of the puerperium.

**Fiskio, P.W.: General Practitioner 11:70 (May) 1955.*

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NEWS AND NOTES

(Continued from page 40)

disability. This points up the need for continuing after-care. The death rate in mental hospitals continued to fall, nearly half of the deaths being in older persons with senile psychoses and the commonest cause arteriosclerotic and degenerative heart disease, with bronchopneumonia second.

—

WINNIPEG CHILD GUIDANCE CLINIC

The second annual report (1954-55) of the operations of the Child Guidance Clinic of Greater Winnipeg has been received. The volume of work continues to increase and an increasing amount of time is being given to screening all referred cases. The clinic now has a maximum of 31 full-time staff, and the Director notes with approval the increasing availability of professionally trained workers. In addition to individual counselling, staff participated in community and educational activities.

Two special surveys were made. The first is referred to as the Cecil Rhodes survey, in which a large school of 619 pupils was studied. Speech, hearing, intelligence and achievement were tested. The clinical staff worked with teachers and obtained a picture of every child in the school. The findings of standardized tests of intelligence and achievement indicated a fairly average picture throughout the school. Findings were discussed with teachers in an attempt to locate children with problems and to plan suitable follow-up programmes for them. From the school 153 children were referred for help to the Clinic. It is notable that in spite of the apparent success of the programme from the clinical point of view, teachers did not consider that it altogether met their needs.

Another report is given of the administration, treatment and academic programme for the special placement class, which is a class for emotionally disturbed children.

—

MEDICAL V.C.'s

This is the centenary year of the Victoria Cross, which was instituted by Queen Victoria on January 29, 1856, as an award for conspicuous bravery. In an article commemorating this centenary, the *British Medical Journal* lists the 38 medical recipients of the Victoria Cross out of the 1,344 awarded. We note that of these doctors three were Canadian. The earliest award to a Canadian was to Dr. H. T. Reade, a surgeon of the 61st Regiment, educated in Quebec and Dublin, who was gazetted in 1861 for bravery in India. The second Canadian recipient was Dr. C. M. Douglas, born in Quebec and educated in Edinburgh, gazetted in 1867. The other Canadian V.C. received his award in World War II. Dr. Scrimger, born in Canada and educated at McGill, was gazetted in 1915 for his bravery at Ypres. He later became Chief Surgeon to the Royal Victoria Hospital, Montreal.

A fourth physician was awarded the V.C. in 1918 for his gallantry while serving in the Canadian

(Continued on page 44)

NEWS AND NOTES

(Continued from page 43)

Army Medical Corps, but he was not a Canadian. This physician, Dr. B. S. Hutcheson, was an American volunteer, who subsequently died in Cairo, Illinois, in 1954.

REPORTING OF NOTIFIABLE DISEASES

The general pattern of reporting of notifiable diseases in Canada is discussed by Dr. E. H. Lossing in the *Canadian Journal of Public Health* (46: 444, 1955). The author is concerned at the low proportion of notifiable cases which are actually reported. From a study of provincial death rates and incidences of certain communicable diseases reported, he finds figures for completeness of reporting which vary from 10.9% in the case of whooping-cough to 17.3% for chickenpox. He suggests that there is an obvious need to simplify reporting procedures and that an educational programme should be begun to acquaint physicians and medical students with the aims of this procedure. He also stresses the valuable data which could be obtained if other types of disability, such as accidents, congenital deformities and certain chronic diseases, were routinely notified.

ASPIRIN AND GASTRIC HÆMORRHAGE

It has been suggested that gastric hæmorrhage is sometimes associated with intake of aspirin, and that patients with gastric lesions should beware of it. A study was therefore made of 151 patients admitted to hospital with hæmatemesis and melæna. Of these 34 never took aspirin, and of the remainder only 28 complained of undesirable side-effects. There was no correlation between the method of taking aspirin and the occurrence of gastric symptoms. Only in three cases was there reasonably good evidence for supposing that the aspirin had caused bleeding, in the absence of any gastric lesion. The authors conclude that serious intolerance occurs in only a small number of patients, and that because of its value as an analgesic, the drug should not be forbidden to patients unless this is really necessary. Those with minor gastrointestinal symptoms from the drug should take a preparation such as calcium aspirin or enteric-coated tablets.—A. P. Waterson, *Brit. M. J.*, 2: 1531, 1955.

CATHOLIC HOSPITAL ASSOCIATION

The Forty-first Annual Convention of the Catholic Hospital Association of the United States and Canada will be held in Milwaukee, Wisconsin, May 21 to 24, 1956. Emphasis in the forthcoming programme will be on the theme: "Education, Research, and Patient Care". Meeting in pre-convention session or simultaneously with the Catholic Hospital Association will be the Conference of Catholic Schools of Nursing, the Purchasing Institute, the Institute for Hospital Dietitians, Conference on Medical Technology, the Institute for Hospital Pharmacists, the Medical Record Library Institute, the Bishops' Representatives, the Chaplains' Conference, and the Institute for X-ray Technicians; there will be a special two-day programme for Hospital Auxiliaries.

THIRD NATIONAL CANCER CONFERENCE

The American Cancer Society and the National Cancer Institute of the Public Health Service, Department of Health, Education, and Welfare, will jointly sponsor the Third National Cancer Conference in Detroit, Michigan, June 4, 5, and 6.

The opening session of the conference, in the Sheraton-Cadillac Hotel, will feature addresses by Dr. John R. Heller, Director of the National Cancer Institute, and Dr. Charles S. Cameron, Medical and Scientific Director of the American Cancer Society. Morning and afternoon sessions of the three-day meeting will begin with a general session, followed by symposia on cancer at different body sites, such as the lung, gastrointestinal tract and breast.

There will be a public Cancer Forum on Tuesday evening, June 5, as a special feature of the Conference. Information may be obtained from the National Cancer Conferences Coordinator, Amer-

(Continued on page 50)

Here's a balanced diet for toddlers that even the busiest mothers can follow!

One of the biggest problems of every mother is time—working according to a strict schedule. And it is often the time factor that prevents a young mother from adhering to a balanced diet for the toddler who has graduated from strained food, but is not yet ready for adult fare.

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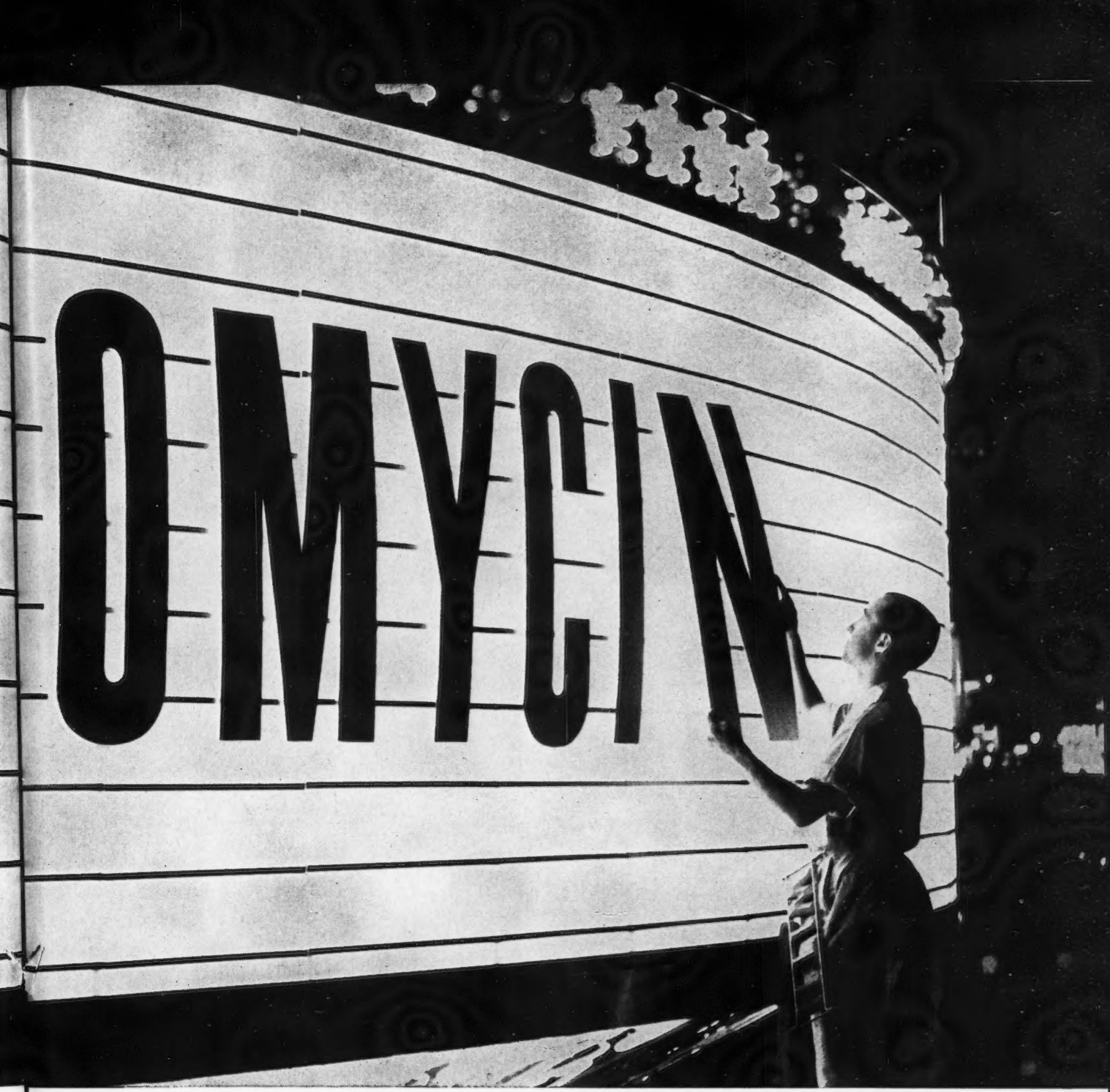

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4) true broad-spectrum activity (proved effective against a wide variety of infections caused by Gram-positive and Gram-negative bacteria, rickettsiae, and certain viruses and protozoa)
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NEWS AND NOTES

(Continued from page 44)

ican Cancer Society, 521 West 57 Street, New York 19, N.Y.

All physicians are invited to attend.

TESTING HYPNOTICS

A recent example of collaboration between general practitioners and university departments is given by the publication of an account of clinical trials of a new hypnotic. Testing hypnotics[1] in a general hospital is not easy because the patient is in strange surroundings and tends to become used to his environment as days go by, and therefore to sleep better; it is more satisfactory to test the effects in the patient's own surroundings. In the trial described, 20 patients of a general practitioner in Sheffield agreed to take part. Three substances were put up in identical form and given to the patient in envelopes designated by a number unknown to the practitioner, and administration was randomized so that equal numbers of patients received the preparations in the six possible orders. One preparation was a placebo, another was cyclobarbitone and the third was alpha-phenyl - alpha - ethyl glutarimide, (glutethimide; Doriden). The object of the investigation was to assess the value of glutethimide, a drug first studied in Switzerland, where it was found to have sedative and hypnotic effects qualitatively similar to those of phenobarbitone, but with a shorter duration of action and an absence of side-effects.[2]

For assessment, ranking methods based on the patients' preference and therefore on subjective criteria were used. Fourteen out of 18 persons put the placebo third in efficacy. Ten chose glutethimide for first place and six cyclobarbitone. It is concluded that glutethimide in a dose of 0.5 g. compares favourably with cyclobarbitone in a dose of 0.2 g. Toxic effects from glutethimide were few (skin rash and nausea). Whether glutethimide is to be given preference to a barbiturate would depend on whether it proves in the long run less toxic and less habit-forming.

1. RUSHBROOKE, M. *et al.*: *Brit. M. J.*, 1: 139, 1956.
2. GROSS, F. *et al.*: *Schweiz. med. Wchnschr.*, 85: 305, 1955.

PROTECTION AGAINST X-RAYS

In 1955 the International Commission on Radiological Protection (I.C.R.P.) issued a 90-page book of recommendations. The topics covered in the report include permissible dose for external and internal radiations, and protection against high energy radiation; one-third of the report is devoted to protection against x-rays.

The recommendations of the I.C.R.P. have been prepared in summary form, and are available in pamphlet form from Radiation Services, Occupational Health Division, Department of National Health and Welfare, Ottawa, Ontario.

The purpose of this document is to emphasize some of the more important aspects of protection connected with the use of diagnostic x-ray equipment, as recommended by the I.C.R.P. It deserves study by all concerned with radiological equipment.

JOURNAL OF ANALYTICAL PSYCHOLOGY

The Society of Analytical Psychologists, London, England, has begun publication of its own journal, called the *Journal of Analytical Psychology*. The journal is published by Tavistock Publications, 2 Beaumont Street, London, W.1. Each volume, which will contain two issues, costs 21 shillings. The journal will particularly interest those educated in the tradition of Jung. In the first issue some of the papers are clinical and others are devoted to the relationship of dreams to primitive thought. There are papers on archetypal themes in depression, counter transference, loathsome women, the father archetype in feminine psychology, and cybernetics and analytical psychology.

STERILIZING SURGICAL GLOVES

A handy 5½" x 11¼" chart entitled "How to Sterilize Surgical Gloves" is available to surgical personnel on writing to the Pioneer Rubber Company, Willard, Ohio. This little chart should prove most useful to hospital staff, and is of a convenient size for hanging on the wall.

Safe, Pleasant Tasting
COUGH SYRUPS
that give
RAPID RELIEF
without
GASTRIC UPSET

Dulsana Compound *dilates bronchi, dulls the cough reflex and renders bronchial secretion, as it occurs, less tenacious without nausea or gastric irritation.* In correct dosage, it is *safe medication* for patients of any age.

"DULSANA" COMPOUND

Indicated at the onset of an acute respiratory infection.
Syrup No. 632 "Frosst"
Each 5 cc. teaspoonful contains:

Carbinoxamine maleate	2 mg. (1/30 gr.)
Ephedrine hydrochloride	4 mg. (1/15 gr.)
Codeine phosphate	10 mg. (1/6 gr.)
Ammonium chloride	100 mg. (1½ gr.)
Chloroform	25 mg. (3/8 gr.)
Menthol	0.25 mg. (1/250 gr.)
Flavoured syrup base	q.s.

Bottles of 3 fluid ounces.

An effective new antihistamine with **bronchodilator, mild sedative** and **local anaesthetic properties.** Drowsiness is unusual.

In the dosage used, **reinforces the bronchodilator action** of the antihistamine.

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Cooling, soothing and mild local analgesics.

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The formula of "Dulsana" Compound WITHOUT CODEINE — especially useful during the exudative stage of an acute respiratory infection.

Bottles of 4 and 8 fluid ounces.

DOSAGE

ADULTS: One or two teaspoonfuls (5-10 cc.) three or four times daily, as required.

CHILDREN, 6-12 years: One-half to one teaspoonful (2.5 - 5 cc.) three or four times daily as required. YOUNGER CHILDREN: Dosage should be adjusted according to age.

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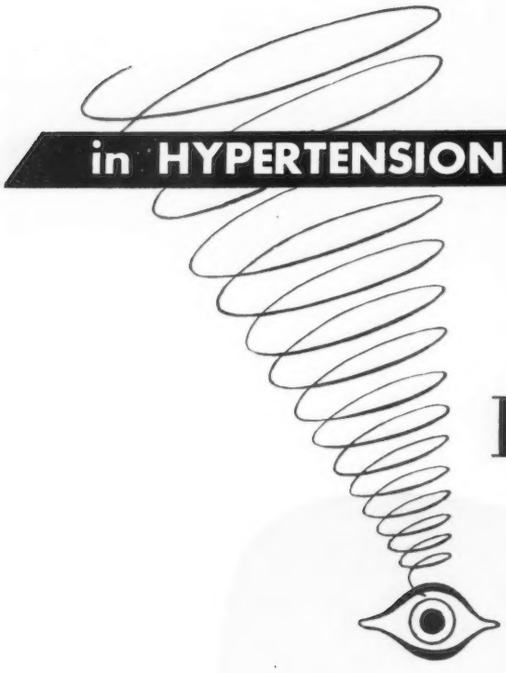

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The results of treatment with Cerutin are not dramatic. It is necessary to adopt a long range viewpoint. Cerutin should be prescribed for every patient with hypertension or diabetes with the hope that capillary rupture in retina and brain may be avoided or postponed.

1. Donegan, J. M. and Thomas, W. A. Capillary fragility and cutaneous lymphatic flow in relation to systemic and retinal vascular manifestations: rutin therapy, Am. J. Ophth., 31:671, 1948.

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to restore appetite and promote weight gain

LACTOFORT

FOR RELUCTANT FEEDERS

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Lactofort improves the protein quality of milk to a point where it approximates that of high-quality meat.

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Vitamin A	3750	I.U.
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Thiamine (as mononitrate)	0.75	mg.
Riboflavin	1.25	mg.
Niacinamide	7.5	mg.
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Folic acid	0.25	mg.
Ascorbic acid (from sodium ascorbate)	75	mg.
Pyridoxine hydrochloride	0.75	mg.
Calcium d-pantothenate	7.5	mg.
Iron ammonium citrate green (elemental iron 7.5 mg.)	50	mg.
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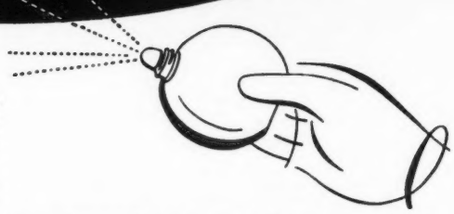

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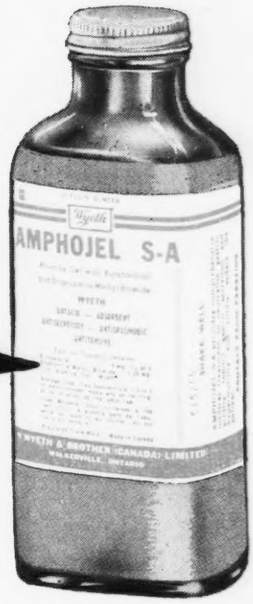

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- controls hypermotility of the gastro-intestinal tract
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Butabarbital..... .16 mg. (¼ gr.)
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AMPHOJEL S-A TABLETS
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Each tablet is the equivalent of one teaspoonful of Amphojel S-A Liquid.

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The comprehensive action of AMPHOJEL S-A offers swift relief for the patient with the vague gastro-intestinal disturbances so common to modern living.

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Although still inconclusive, laboratory evidence and extensive clinical studies in Europe and Canada suggest the following mechanism operates. Stilbestrol diphosphate sodium is essentially a "transport form" of stilbestrol. The compound is water-soluble, well-tolerated, and pharmacologically inert at injection. It becomes activated preferentially in malignant prostatic tissue because of the high level of phosphatase activity there. On reaching the prostate, the phosphate groups are removed from the Honvol molecule by the enzyme phosphatase, and active stilbestrol is released directly in the tumour and its metastases.

Such localization of high stilbestrol concentrations specifically in malignant prostatic tissue makes possible a degree of cytotoxic action unobtainable with other hormone therapy.

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This release of stilbestrol presumably can take place wherever phosphatases are present. But the uniquely high levels of this enzyme in carcinatomous prostatic tissue assure target selectivity with virtual absence of systemic hormonal effects. This has certainly been borne out in the clinical studies *where feminizing effects have been rare and minor.*

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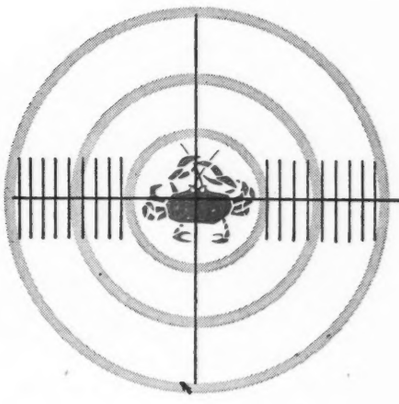
For intravenous use only.

CLINICAL RESULTS

The results of clinical studies are too extensive for outline here. They are available from Frank W. Horner Limited. But it can be said that the rapid improvement of prostatic carcinoma —both clinically and biochemically—with negligible side effects—makes Honvol therapy very heartening. In particular, it offers real hope in those cases which have become refractory to castration and present-day hormone control methods.



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conform bandage
• clings to itself • elastic • conforms easily
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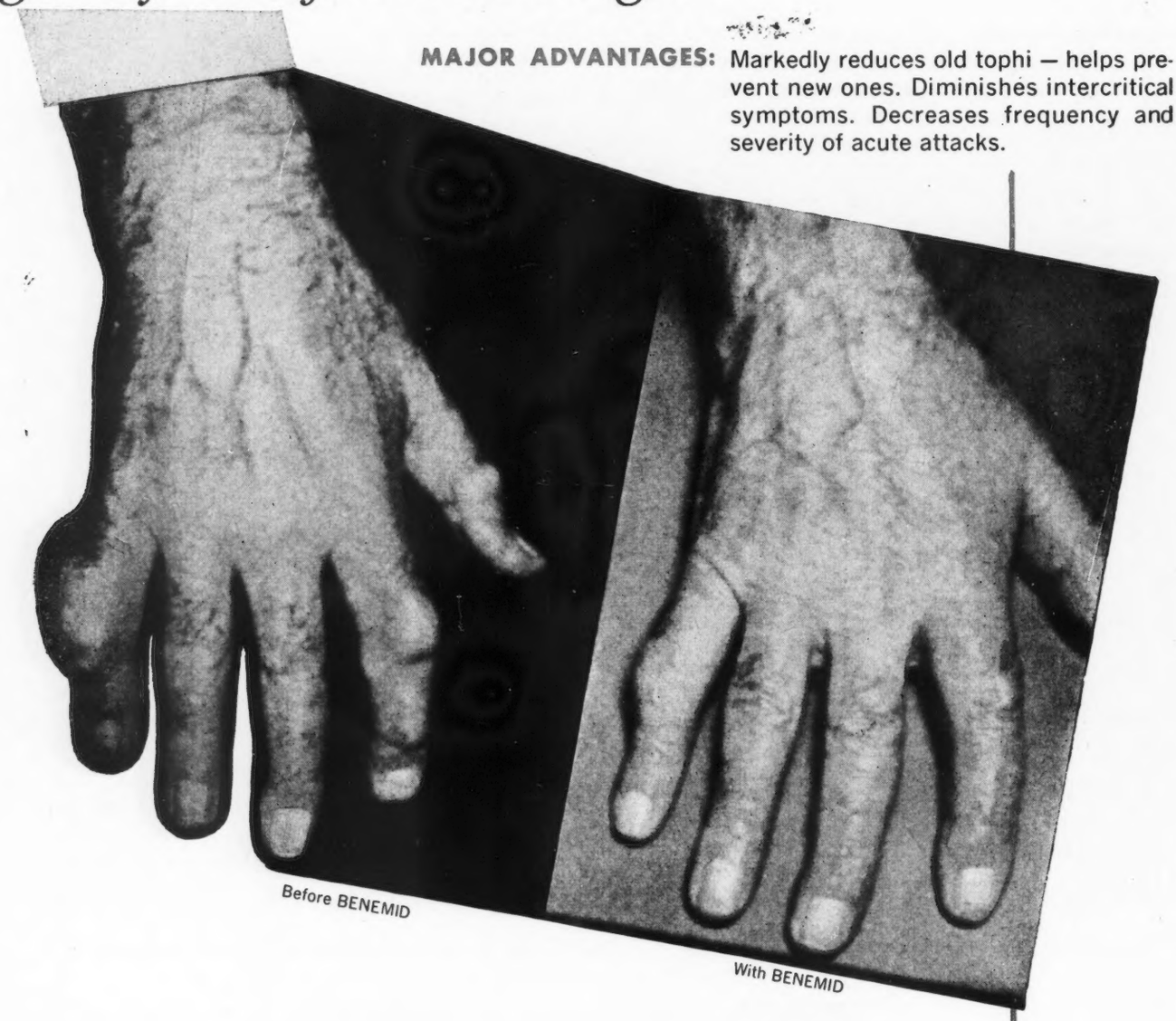
INDEX TO ADVERTISERS

'Benemid'

PROBENECID

greatly benefits chronic gout

MAJOR ADVANTAGES: Markedly reduces old tophi — helps prevent new ones. Diminishes intercritical symptoms. Decreases frequency and severity of acute attacks.



Many patients on BENEMID therapy return to useful occupations, even those requiring skilled use of the hands.

"Apparently the most . . . harmless uricosuric agent,"[1] BENEMID tends to restore and maintain almost indefinitely normal serum uric acid levels.

Urate deposits in the joints are thus reduced . . . new ones prevented. Further, the "periodic administration of BENEMID may reduce the need for surgical interference."[2]

Supplied: in 0.5 *Gm.* tablets. *Dosage* 1 to 4 tablets daily.

Note: Salicylates should not be used with BENEMID since these drugs inhibit each other's action.

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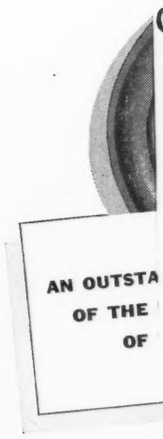
References: 1. J.A.M.A. 149:1188, 1952. 2. Gout and Gouty Arthritis, Modern Medical Monographs, 7, New York, Grune & Stratton, 1953, p. 80.



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